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PROCEEDINGS
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*THE SPECTRUM OF CAPELLA**

By Otto Struve

Berkeley Astronomical Department, University of California

Read before the Academy, April 25, 1951

1. The spectrum of Capella (alpha Aurigae) shows many unusual features, some of which have not been recorded previously in other stars. In several respects this is the most baffling astronomical spectrum which I have ever observed.

2. Capella, the second brightest star of the northern sky, is a binary consisting of two giant suns which are of approximately the same brightness in visual light. One component is yellow, like the sun, the other is considerably bluer. Hence, in infra-red light the continuous spectrum is almost entirely that of the yellow, G-type star. In the ultra-violet, at about λ 3700, the light of the bluish, F-type star predominates. J. A. Anderson and P. W. Merrill have measured Capella as a visual binary with the great Michelson interferometer of the Mount Wilson Observatory. Hence, there can be no doubt as to the duplicity of the star. The period is 104 days, and the orbit is nearly circular. The inclination is 41 degrees, and there are no eclipses. The components are about 10 and 8 times larger than the sun, in diameter, and have about 4 and 3 times the mass of the sun, respectively, for the G and F components. The distance between their centers (127×10^6 km.) is nearly ten times the sum of their radii. The velocity curve of the G component has been accurately determined by several observers, but the radial velocities of the F component are quite uncertain. A mass ratio of G/F = 1.2 has usually been adopted. The distance of Capella is very accurately known from the combination of the interferometric orbit (which gives its size in angular measure) and the spectrographic orbit (which gives the same quantity in kilometers). Because of this circumstance Capella has played an exceptionally important role in the derivation of Eddington's mass-luminosity relation: the masses are individually determined if the mass-ratio is known, and the absolute visual magnitude of each component is about zero (nearly 100 times more luminous than the sun). Although the photoelectric observations by J.

Stebbins have shown that there are no eclipses, P. Guthnick has found irregular oscillations in the combined brightnesses of the two stars amounting to about 0.1 mag.

3. The combined spectrum shows a large number of absorption lines. Nearly all the fainter ones can be identified with the G star. These lines show no conspicuous change with phase. They are absent in the light of the F star. Hence, they are overlaid by the continuous spectrum of the latter, and appear shallow in the ultra-violet and the violet, but of normal depth in the red and infra-red.

4. Many absorption lines which are normally strong in a solar-type star are, at certain times, absent or very weak in the G component. These same lines are usually present in the F component—as very broad, diffuse and shallow lines resembling those produced by large statistical Doppler effects. It is still impossible to explain the resulting profiles: they suggest rapid rotation in the case of the Na I lines, but the strong Fe I lines in the ultra-violet are too deep. Perhaps the best preliminary explanation is that they are produced by large turbulence (most probable velocity about 30 km./sec.) and that in the visual and infra-red regions the continuous light of the G star veils the normally deep profiles.

5. The most remarkable feature is the absence or weakness in the G star of those lines which are strong, moderate or even quite weak in the F star. This is pronounced at the elongations, especially when the G star is approaching. At the conjunctions the spectrum is normal (it resembles alpha Bootis when the G star is in front, but shows a tendency in the direction of the F supergiant alpha Persei when the F star is in front). At both conjunctions many lines which are strong in alpha Bootis and alpha Persei are also strong in Capella—stronger, in fact, than the sum of the corresponding two components observed at the elongations. The weakness of the strongest Fe I lines in the G star at elongations is not connected with excitation potential or multiplet structure. When a strong and a weak line have common lower or upper levels the strong line alone is affected and is sometimes rendered much fainter than the normally weak line. This effect is not photographic or instrumental. It has nothing to do with the populations of the various atomic levels, and it does not depend upon the exciting or ionizing action of the light of the F star. Whenever a strong line is missing in the G component, the corresponding space of continuous spectrum looks entirely smooth. Whenever a weakened G-line remains, its profile gives the impression of being broadened by damping in the manner expected for a strong line. The profile then looks as though it were veiled by atomic emission which fills in most of the line depth. This phenomenon is extraordinarily conspicuous in Fe I, Al I, Mg I. It is present, but less conspicuous in Ca I, Sr II, Ti II. But the Na I lines remain unaffected in the G star.

A few examples will illustrate these effects: (*a*) Fe I multiplet 43: a^3F–y^3F^o, e.p. 1.5 volts. The strong lines λλ 4046, 4064, 4072, 4005 are greatly weakened in the G star during elongations and are present in the F star. But the weak line Fe I 4032.6, Mult. 44, a^3F–y^5P^o, e.p. 1.5 volts, is quite normal. (*b*) Fe I multiplet 42: a^3F–z^3G^o, e.p. 1.5 volts. The strong lines λλ 4272, 4308, etc., are greatly weakened in the G star during elongations; but the weak line Fe I 4148, of the same multiplet, is not affected. (*c*) All very strong Fe I lines of low e.p., arising from levels a^5D and a^5F, are almost extinguished in the G star during elongations and are strong in the F star, but Fe I 3906.5, Mult. 4, a^5D–z^5D^o, e.p. 0.1 volt, does not vary with phase. The only pronounced effect of excitation potential is that *all* lines arising from low atomic levels of Fe I are systematically much weaker in Capella than a α Bootis. This could be explained as the result of a difference in the excitation temperatures of these G and K giants. (*d*) Fe I lines of high e.p. show no variation with phase, and no systematic weakening (other than that caused by the filling in of the F continuous spectrum). Thus, Fe I 3986.2, 4040.6 (Mult. 655, a^3D–v^3F^o; e.p. 3.3 volts) are perfectly normal. (*e*) Fe I 3795.0 (Mult. 21: a^5F–y^5F^o, e.p. 1.0 volt) is normally a fairly strong transition, and is greatly weakened in the G star during elongations. It is weak and diffuse in the F star. But the neighboring, normally weak, transition Fe I 3794.3 (Mult. 177: a^3H–z^3I^o, e.p. 2.4 volts) is entirely normal. (*f*) Fe I 4216.2 (Mult. 3: a^5D–z^7P^o, e.p. 0.0 volt) is close to the line Sr II 4215.5 which is affected in the G star and is present in the F star. The Fe I line undergoes changes that we tentatively attribute to the varying amount of veiling by the continuous spectrum of the F star. It is normally a weak transition, and is not expected to show in the F star. Another, equally weak member of the same Fe I multiplet, λ 4206.7 is entirely normal.

6. The spectrum of the F star is abnormal. It shows only the strongest lines of Fe I, and the "gradient" of total absorptions within each multiplet is probably steeper than is required by turbulent Doppler broadening (total absorption is proportional to number of atoms in the lower level). The H lines are much too weak for an F giant: Hβ, γ, δ are present as weak, hazy absorptions; the higher members are not visible. Hα is almost absent because of the relative weakness of the F star's continuous spectrum in the red.

7. The very strong Ca II absorption lines H and K are much shallower than can be explained as a result of the superposition of two normal components corresponding to types G and F. The G star has a centrally reversed emission core, with appreciable turbulent broadening (v_t ~30 km./sec. in the chromosphere of the G star; in the G reversing layer v_t ~5 km./sec.). The blended absorption line shifts with respect to the emission, but is roughly undisplaced with respect to the center of mass.

Yet, the deep and broad profiles of normal F and G stars would add up to produce a profile that is not very different from the line in either α Bootis or in α Persei, alone. It is probable that the centrally reversed emission line is only the peak of a rather broad emission profile which fills in much of the normal absorption profile. According to this tentative hypothesis it is tempting to attribute the filling in of the strong lines of Fe I to similar emission profiles. The emitting atoms must then come from great heights above the reversing layer of the G star. Perhaps they have the character of prominences or flares, or swarms of particles, such as give rise to the aurora borealis in the earth's upper atmosphere.

But this effect does not fully explain the behavior of the strong lines of Fe I, etc., in the G star, as was described in section 5. The strong infrared Ca II multiplet $\lambda\lambda$ 8498, 8542, 8662 (3^2D–4^2P^o) is not appreciably contaminated by any central emission, nor is there any general filling in of the line profile. We find little filling in of the Na I lines D1 and D2, or of Hα. The metallic lines are not strong enough to be certain of this point; in the ordinary red region they are all appreciably weaker than in α Bootis. There is thus a suspicion that somehow the weakening of the strong lines in the G star is connected with the presence of the F star's spectrum. Without implying a physical explanation it seems as though an observable trace of an F absorption line is required to extinguish the corresponding line in the G star.

Sometimes a faint, unrelated Fe I line falls very close to the position of the vanished G type line. When that happens the unrelated line is not blotted out. Although it sometimes undergoes large changes in apparent intensity, these can usually be explained in terms of varying amounts of continuous light from the F star.

8. The centrally reversed emission cores of Ca II H and K of the G star are present at all phases, and their profiles are usually symmetrical. But at phases 57 to 61 days (F star in front) the violet half was about three times more intense than the red: V/R = 3. At phase 64.7 days the symmetry has disappeared. It is not yet certain that this variation is periodic.

9. The Hα absorption line is strong and belongs to the G star (on a few plates there may be a very faint, broad absorption feature corresponding to the F star). It is conspicuously unsymmetrical. At phase 30 days (G star receding) the line is shaded toward the longer wave lengths. At phase 60 days (F star in front) it is slightly shaded in the opposite direction. This feature is not necessarily connected with the anomalous intensities of the strong lines of Fe I, etc.

10. The velocity curve of the F component can only be determined with great uncertainty. The lines of this component are unusually broad. In the blue, green and yellow regions they are filled in by the continuous spectrum of the G star. The only well-defined pairs of lines are those of

Na I, D_1 and D_2. But the F lines are very shallow and at their edges overlap with the narrow lines of the G component. Hence their measurements are not very reliable. At phase 30.8 days the separation G − F = 50.6 km./sec. Using a circular orbit by W. Struve, with K_G = 26.0 km./sec.; K_F = 31.1 km., and phase zero corresponding to that conjunction at which the G star is in front, we find an expected separation of

$$\Delta v = (K_G + K_F) \sin 360° \times \frac{30.8}{104} = 54.7 \text{ km./sec.}$$

The agreement is reasonably good, and confirms the *approximate* value of the mass ratio found by previous observers. But it should be emphasized that these observers had no knowledge of the peculiar profiles and intensities of the F lines. They probably measured, in effect, sharp lines of the G star which appear considerably enhanced, photographically, when they accidentally fall within a broad absorption line of the F star. Individually, such measurements must have yielded poor results (as indeed all previous observers have commented upon), but statistically a reasonably accurate velocity would result.

In the ultra-violet and photographic regions I measured by means of a differential procedure several displacements of the type

$$(K_G + K_F)\,(\sin [\text{phase } 1] - \sin [\text{phase } 2])$$

The two phases were 30 days and 83 days, respectively. The formula predicts a total displacement of 110.1 km./sec. My measurements show a dependence upon the intensities of the F lines:

	TOTAL DISPLACEMENT, KM./SEC.
Strong Fe I lines near λ 3770	85
Very strong Fe I, Mg I near λ 3825	99
Moderately strong lines of Fe I, Sr II, Hδ near λ 4000	62

Until we know whether the epoch and period used in computing the phases are still correct, the systematic departure of our measurements from the accepted value need not necessarily be attributed to an error in the accepted mass ratio. But the large range in the observed displacements is undoubtedly real: it can be easily seen when two spectrograms at opposite phases are laid side by side, with the numerous faint lines of the G star brought into coincidence. This result would seem to indicate that the broadening effect of the F lines is not symmetrical; in other words, these lines are not produced in an ordinary reversing layer, but in a turbulent stream connected with the F star, but having an unsymmetrical distribution of velocities with respect to the latter's center of mass.

11. The intensities of the F lines vary with phase. They are stronger near phase 83 days (F star receding) than near phase 30 days. Changes of

this kind have been observed in several spectroscopic binaries, but usually the approaching component has the stronger absorption lines (α Virginis).

12. A few lines of the blended spectrum exhibit extraordinary changes with phase that are difficult (but perhaps not impossible) to explain by a combination of the various effects considered separately in the preceding paragraphs. A remarkable example is Fe I 4271.8. At phase 83 days it is exceedingly strong, and much of the combined intensity is undoubtedly due to the F line. But at phases 25–30 days there is only a weak trace of the G star's Fe I 4271.8. The neighboring G line Fe I 4271.2 is enhanced at the latter phase, but there is almost nothing left of the F line. As a rule the difference in the intensities of the F lines at the two elongations is not nearly as large. Hence, even this variation is itself different for different lines!

13. Although there is still some uncertainty concerning the phases of the spectrograms used in section 10, and the mass ratio $\alpha = \mathfrak{M}_1/\mathfrak{M}_2$ is not determined, we already know that different lines yield different α's. Hence, it is of interest to appraise the possible influence of an error in the presently adopted value $\alpha = 1.20$ upon the masses of the two components. Assuming again a circular orbit with the well-determined values $P = 104.023$ days, $K_1 = 26.05$ km./sec., we find

$$f(\mathfrak{M}) = \frac{\mathfrak{M}_2}{(1+\alpha)^2}\sin^3 i = [3.0164\text{–}10]PK_1^3 = 0.191\odot$$

The usual assumption, $\alpha = 1.2$, gives $\mathfrak{M}_1 = 3.9\odot$, $\mathfrak{M}_2 = 3.2\odot$. It is entirely possible, however, that $\alpha = 1.0$. This would give us $\mathfrak{M}_1 = \mathfrak{M}_2 = 2.7\odot$. The difference is sufficient to produce a marked shift in the mass-luminosity diagram.

14. The spectroscopic anomalies of Capella are not observed in single stars. They must be the result of an interaction between the components of this binary system. It was previously believed that in a pair, whose separation exceeds by a factor of 10 the sum of its radii, this interaction may be neglected. But the components of Capella are giant stars, having mean densities which are less than 0.01 of the mean density of the sun. It is, therefore, not surprising that large interactions are present in the outermost layers of both components.

* This investigation was made possible by a cooperative arrangement between the Berkeley Astronomical Department of the University of California and the Mount Wilson and Palomar Observatories.

ON THE STRUCTURE OF JERVINE: DIACETYL-7-KETOJERVINE

BY O. WINTERSTEINER, M. MOORE, J. FRIED AND B. ISELIN

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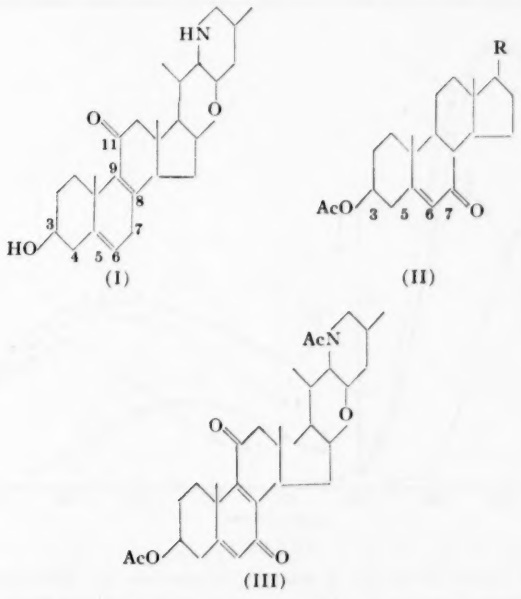
Communicated March 13, 1951

The constitution of jervine, the main alkaloid of *Veratrum viride*, has been the subject of several important communications by Jacobs and his collaborators,[1-3] in the latest of which[3] this steroidal base is formulated



(I) (II) (III)

as I. The presence of a keto group adjacent to an $\alpha,\alpha,\beta,\beta$-substituted double bond was deduced from the ultra-violet absorption spectrum (maxima at 250 mμ, ϵ 15,000, and at 360 mμ, ϵ 60), and its site at the hindered position 11 from its inertness toward ketone reagents and catalytically activated hydrogen. The adjacent double bond had then by necessity to be accommodated in the 8,9-position, since the other two available positions β to the carbonyl are occupied by quaternary carbon atoms. While thus the evidence for the location of the α,β-unsaturated keto grouping is purely circumstantial, the fact that such a group exists in jervine is well substantiated by chemical findings which need not be elaborated here. Likewise, there can be little doubt from the work of Jacobs with Huebner[1]

and with Sato[2] that the positions of the hydroxyl group (C_3) and of the other, isolated, double bond (5,6) have been correctly assigned.

This formulation of jervine places the alkaloid in close relation to cortisone and other adrenocortical steroids carrying oxygen in position 11, and suggests the possibility of its use as a starting material for the synthesis of this hormone. With this latter point in mind we have since some time been engaged in attempts to adduce more decisive evidence for the location of the unreactive keto group. These studies have now progressed to a point where it seems desirable to communicate some of the results. We are

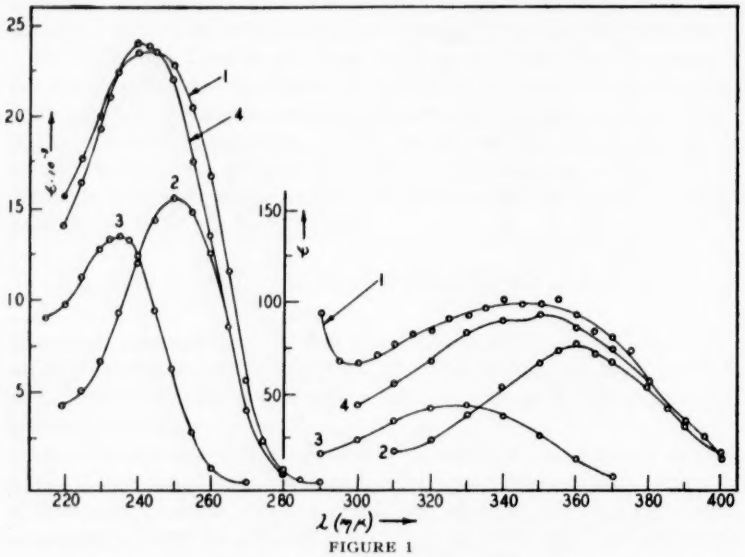


FIGURE 1

Ultra-violet absorption spectra of diacetyl-7-ketojervine (*1*), diacetyljervine (*2*), and 7-ketocholesteryl acetate (*3*); (*4*): composite curve calculated from (*2*) and (*3*). Solvent: absolute ethanol.

reporting here first on a new derivative of jervine, already obtained by us two years ago, the properties of which are difficult to reconcile with the above structural concept. Its preparation was suggested by the following simple consideration:

Cholesteryl acetate and other steroids possessing an isolated 5,6-double bond can be converted by means of chromic acid into the corresponding 7-ketones (II). It might be expected that in jervine as formulated by Jacobs (I) the methylene group in position 7, being activated by two adjacent double bonds, should be even more susceptible to allylic oxidation than in

ordinary Δ^5-stenols. On this premise diacetyljervine should yield with chromic acid a diketone of structure III (or products derived from III by further oxidation). The insertion of the new keto group would extend conjugation to position 5 and hence should produce a marked red shift in the ultra-violet absorption spectrum. A diene-dione system identical with that in III occurs in acetoxy-lanostadiendione,[4] a derivative of the triterpene lanosterol; the spectrum of this compound exhibits a high maximum at 275 mμ (log ϵ 4.2).

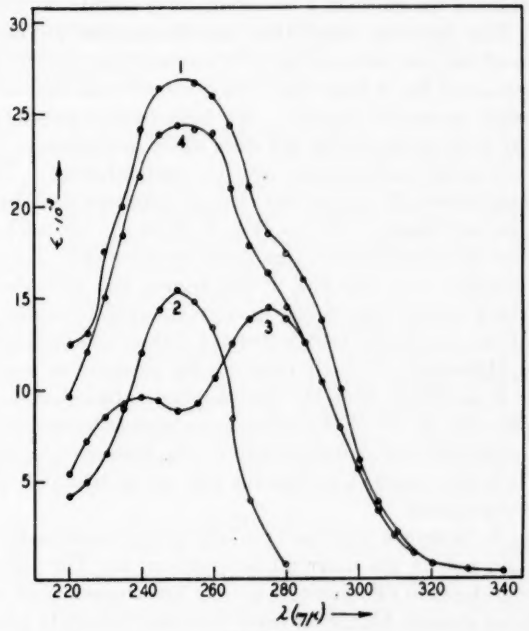


FIGURE 2

Ultra-violet absorption spectra of diacetyl-7-ketojervine semicarbazone (*1*), diacetyljervine (*2*), and 7-ketocholesteryl acetate semicarbazone (*3*); (*4*): composite curve calculated from (*2*) and (*3*). Solvent: absolute ethanol.

We have subjected diacetyljervine to oxidation with chromic acid under a variety of conditions, and in every case obtained the expected ketone, diacetyl-7-ketojervine (m.-p. 237–239°, $[\alpha]_D^{24} - 166°$), in 7–10% yield. It readily formed a monosemicarbazone and a mono-2,4-dinitrophenylhydrazone. Careful saponification with potassium carbonate or potassium bi-

carbonate in 90% aqueous methanol effected removal of the O-acetyl group, thus affording N-acetyl-7-ketojervine (m.-p. 262–264°, $[\alpha]_D^{23}$ −192°).

Figure 1 gives the ultra-violet absorption curve of diacetyl-7-ketojervine together with those of diacetyl jervine and of 7-ketocholesteryl acetate. There is no sign of the red shift expected on the basis of structure I. To the contrary, the main band is displaced from its position in the jervine spectrum to a somewhat lower wave length (245 mμ), and its height is much increased. It is furthermore apparent from figure 1 that the experimental curve (curve 1) closely approximates one (curve 4) which is obtained by superimposition of the curve of 7-ketocholesteryl acetate on that of diacetyljervine. This can only mean that the chromophor produced by the introduction of the new keto group (Δ^5-7-carbonyl) is not in conjugation with, but separated by at least one carbon atom from, the chromophoric group originally present in jervine. We have further substantiated this conclusion by making use of the red shift which accompanies the conversion of an α,β-unsaturated ketone into its semicarbazone. Thus in the case of 7-ketocholesteryl acetate this change manifests itself by the emergence of a new high band at 275 mμ (Fig. 2, curve 3). It can be seen that in the curve of diacetyl-7-ketojervine semicarbazone (Fig. 2, curve 1) this band is in evidence as a shoulder in this region, and that the curve can again be arrived at with a fair degree of approximation by adding the ordinate values of the curves of 7-ketocholesteryl acetate and of diacetyljervine. At any rate, this result definitely rules out the presence of a system such as II, since it is known that the chromophor —HC=CH·C:(N·NH·CO·NH_2)CH=CH in $\Delta^{1,4}$-cholestadien-3-one semicarbazone, even though it lacks the additional keto group present in III, gives rise to a high band at 300 mμ.[5] It is thus quite clear that the two α,β-unsaturated keto groupings are not contiguous.

It remains to be shown that the new keto group has actually entered at position 7 and not at the other allylic position, 4. The following facts should be considered in this connection: (1) The chromic acid oxidation of Δ^5-unsaturated steroids has never been observed to lead to products substituted with oxygen in position 4; the only positions affected are 5, 6 and 7. (2) The maximum at 241 mμ in the spectrum of Δ^5 cholesten-4-one[6] shows a much lower extinction (ϵ = 7200) than that at 235 mμ in the spectrum of 7-keto-Δ^5-stenols (ϵ about 14,000). The high extinction coefficient (24,000) of the composite maximum in curve 1, figure 1 (diacetyl-7-ketojervine) is not explicable on this basis. (3) Diacetyldihydrojervine (which differs from diacetyljervine by the absence of the double bond adjacent to the unreactive keto group) on oxidation with chromic acid yielded a ketone (m.-p. 281–284°, $[\alpha]_D$ −116°) analogous to that obtained from diacetyl jervine. In this case there can be no doubt from the ultra-violet absorption characteristics (maxima at 233 mμ, ϵ = 15,300, and at 310 mμ,

$\epsilon = 80$) that the new keto group has entered position 7. (4) N-acetyl-7-ketojervine proved to be inert toward periodic acid. If the new keto group were in position 4, this oxidant should have attacked the 4,3-ketol grouping with the formation of the *seco*-3-aldehydo-4-carboxylic acid.

The molecular rotation changes incident to the formation of the two new ketones are likewise in agreement with the Δ^5-7-keto structure assigned to them (table 1). However, it should be mentioned that the molecular rotation difference $[M]_D$ diacetyl-7-ketojervine $-$ $[M]_D$ N-acetyl-7-ketojervine, namely $-870° + 925° = +55°$, is considerably more positive than the usual value for the change Δ^5-stenol $\rightarrow$ Δ^5-stenyl acetate ($-35°$).[7] On the other hand, with jervine and 7-ketocholesterol the molecular rotation differences for this change fall within the accepted range of experimental error (N-acetyljervine $\rightarrow$ diacetyljervine: $-582° + 555° = -27°$; 7-ketocholesterol $\rightarrow$ 7-ketocholesteryl acetate: $-455° + 416° = -39°$).

TABLE 1

	Δ^5-STENYL $[M]_D$	Δ^5-7-KETO-STENYL $[M]_D$	$\Delta[M]_D$
Cholesteryl acetate	$-184°$	$-455°$	$-271°$
Diacetyljervine	$-582°$	$-870°$	$-288°$
Diacetyldihydrojervine	$-317°$	$-610°$	$-293°$

Nevertheless, the evidence is overwhelmingly in favor of the 7-keto-structure of the new compounds. It then follows from the ultra-violet absorption data discussed further above that jervine cannot possess a double bond in the 8,9-position (or for that matter, in the 8,14-position), and further that its unreactive keto group cannot occupy the 11-position, unless it be assumed that there is some abnormality in the architecture of rings C or D of the steroid skeleton. We shall present evidence in a future communication that such is probably the case.

Experimental.—All melting points are corrected for stem exposure. The specific rotations were measured in chloroform unless indicated otherwise.

Diacetyl-7-ketojervine: Diacetyljervine (966 mg., 1.9 millimole) was dissolved in pure acetic acid (10 cc.). Chromium trioxide (635 mg., 5×1.9 milliatoms of oxygen) was added in small portions to the mechanically stirred solution in the course of 90 minutes while the temperature was maintained at 55°. Stirring was continued at that temperature for two more hours. After cooling, the excess chromic acid was destroyed by the addition of a small volume of methanol. The solution was poured into ice-cold water, and the mixture was extracted with chloroform (3×200 cc.). The chloroform solution was washed with potassium carbonate solution and water, dried [redacted] and evaporated to dryness (705 mg.). (The amorphous acidic products (158 mg.) recovered from the carbonate extract were not further investigated.) The neutral fraction was treated with Girard's reagent T (700 mg.) in absolute ethanol (15 cc.)–acetic acid (1.5

cc.) by boiling the solution for 20 minutes and then allowing it to stand at room temperature for 20 hrs. The mixture was poured into ice water containing potassium hydroxide equivalent to 90% of the acetic acid present. After removal of the non-ketonic products by extraction with ether (3 × 100 cc.) the aqueous phase was acidified to pH 1 with hydrochloric acid. Within a short time it began to deposit crystals which after 2 hrs. were filtered off and washed with water (94 mg., m.-p. 220–223° dec.). Two recrystallizations from absolute ethanol yielded rosettes of small needles melting at 237–239° (67 mg.), $[\alpha]_D^{23}$ −166° ±1° (*c*, 1.029); −164° ± 2° (*c*, 0.514 in ethanol).

Anal. Calcd. for $C_{31}H_{41}O_6N$ (523.6): C, 71.10; H, 7.89; N, 2.68. Found: C, 70.98; H, 7.84; N, 2.62.

A few comments on the procedure used might be appended: Chloroform cannot be employed for the extraction of the non-ketonic material, as the Girard derivative of the ketone passes into this solvent at pH 6. The ketonic material (51 mg. in the above experiment) obtained by ether extraction of the filtrate from the crystalline precipitate could in no case be induced to crystallize. The non-ketonic fraction was always found to contain considerable amounts of unattacked starting material recoverable by crystallization from 50% aqueous ethanol. Variations of the above oxidation procedure (standing at room temperature for 1–3 days, use of sodium chromate in acetic anhydride–acetic acid[8]) were explored, but the yields were generally somewhat lower than in the experiment described.

The *semicarbazone* was prepared by treating the ketone with excess semicarbazide acetate in 90% ethanol for 2 hrs. at reflux temperature. It formed tiny needles from ethanol-water, m.-p. 245–255° (dec.).

Anal. Calcd. for $C_{32}H_{44}O_6N_4$ (580.7): N, 9.65. Found: N, 9.48.

The *2,4-dinitrophenylhydrazone* was prepared in the usual manner (room temperature) with Brady's reagent. Orange-colored needles from benzene-ethanol, m.-p. 293–298° (dec.), $\lambda_{max.}^{alc.}$ 252 mμ (ε 19,200), 372 mμ (ε 18,100).

Anal. Calcd. for $C_{37}H_{46}O_9N_5$ (694.8): C, 63.96; H, 6.53; N, 10.08. Found: C, 63.21; H, 6.23; N, 9.99.

N-acetyl-7-ketojervine: Since diacetyl-7-ketojervine apparently undergoes deep-seated structural changes in strongly alkaline solution (see below), potassium carbonate or bicarbonate had to be used for the removal of the O-acetyl group.

To a solution of diacetyl-7-ketojervine (31.4 mg.) in methanol (3 cc.), potassium carbonate (14.3 mg.) dissolved in 0.3 cc. of water was added. The solution, which turned orange in about 10 minutes, was allowed to stand under nitrogen gas at room temperature for 28 hrs. It was concentrated to a small volume in a nitrogen current, diluted with water, and acidified to congo blue with 10% acetic acid. The crystalline product, part of which had formed already during the removal of the methanol, was centri-

fuged and washed with water (28 mg., m.-p. 241–250°). It was dissolved in benzene, and chromatographed on a small column (6 × 30 mm.) of sulfuric acid-washed alumina. Continued washing with benzene removed small amounts of a yellow oil, whereupon the crystalline product was eluted with benzene-ether 9:1. Two crystallizations from small volumes of acetone yielded the pure N-acetate in form of small needles m.-p. 262–264°, $[\alpha]_D^{23}$ −192° ± 2° (*c*, 0.779, 1 dm.); $\lambda_{max.}^{alc.}$ 247 mμ (ε 24,300): 343 mμ (ε 97).

Anal. Calcd. for $C_{29}H_{39}O_5N$ (481.6): C, 72.32; H, 8.16. Found: C, 72.20; H, 8.20.

In a simultaneous experiment a mixture of 30 mg. each of the diacetate and of potassium bicarbonate, partly dissolved in 3.75 cc. of 80% aqueous methanol, was shaken in a glass-stoppered vessel for 40 hrs. The faintly yellow solution which still contained a small amount of undissolved material was worked up as described above. Chromatographic purification followed by recrystallization from acetone yielded 10 mg. melting at 263–265°.

Diacetyl-7-ketojervine dissolves in methanolic potassium hydroxide forming an intensely orange solution which very soon turns deep red. Formation of an enolate is evidently involved, as dilution with water fails to produce a precipitate. On prolonged standing irreversible changes occur, since only yellow oily products could be isolated after acidification. Neither diacetyl jervine nor 7-ketocholesteryl acetate give a halochrome reaction with alkali. It would seem therefore that the two chromophors are separated by no more than two carbon atoms. This matter will be further investigated.

Diacetyl-7-ketodihydrojervine: Diacetyl dihydrojervine (421 mg., 0.82 millimole) was dissolved in a mixture of acetic acid (50 cc.) and benzene (17 cc.). A solution of chromium trioxide (287 mg., 5.25 × 0.82 milliatoms of oxygen) dissolved in a few drops of water and 3 cc. of acetic acid was added dropwise. After standing for two days at room temperature excess chromic acid was destroyed by adding a small volume of ethanol, the green solution was concentrated almost to dryness *in vacuo*, and the residue was worked up as described in the section on diacetyl-7-ketojervine, except that ether instead of chloroform was used. The neutral products (161 mg.) were treated with Girard's reagent T at room temperature for two days. The aqueous phase containing the ketone hydrazone on acidification deposited a crystalline precipitate (64 mg., m.-p. 264–273°) which was recrystallized several times from absolute ethanol. Small square plates, m.-p. 281–284° $[c]_D^{25}$ −116° ± 1° (*c*, 0.922, 1 dm.); $\lambda_{max.}^{alc.}$ 233 mμ (ε 15,300), 310 mμ (ε 80).

Anal. Calcd. for $C_{31}H_{43}O_6N$ (525.7): C, 70.84; H, 8.25. Found: C, 70.73; H, 8.43.

A solution of the compound in 5% methanolic potassium hydroxide remained colorless.

Acknowledgment.—The authors are indebted to Mr. J. F. Alicino and his assistants for the microanalyses, and to Dr. Nettie Coy for the ultra-violet absorption data.

Summary.—Diacetyl-7-ketojervine has been prepared by chromic acid oxidation of diacetyljervine. The ultra-violet absorption characteristics of this compound and of its monosemicarbazone indicate that the double bond adjacent to the unreactive keto group in jervine cannot occupy the 8,9-position as postulated by Jacobs and his collaborators. Hence the site of this keto group cannot be carbon atom 11, unless it be assumed that there exists some structural abnormality in rings C or D of the steroid nucleus.

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*THE EFFECT OF OXYGEN ON MUTATION INDUCTION BY X-RAYS**

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Communicated by G. W. Beadle, March 6, 1951

Interest has recently been revived in the observation that the biological effects of x-rays are greatly influenced by the oxygen tension of the medium in which the material is maintained during irradiation. A striking decrease in irradiation damage resulting from low oxygen tension during exposure to ionizing irradiation has been reported for tumor cells,[1] bean root tips,[2] yeast[3] and mammals.[4, 5] X-ray-induced chromosome aberrations in *Vicia faba* root tips,[6] barley seeds,[7] *Tradescantia* microspores,[8] and mutations in Drosophila[9] are greatly decreased when irradiation is carried out under low oxygen tension. Hollaender and coworkers[10] found that the survival ratio of *Escherichia coli* is also strikingly reduced when suspensions are exposed to x-rays in an atmosphere of oxygen as compared to air, and greatly increased when irradiated in nitrogen. It is possible to detect and assay certain biochemical mutations of bacteria, even when they occur

at very low frequencies, by taking advantage of the specific growth requirements of the mutant or of its back mutation. Therefore, it was considered feasible to determine whether a change in oxygen tension would alter the induction of bacterial mutations by x-rays. The work here presented is part of a study being conducted as a group project in this division on factors modifying the biological effects of x-rays.

Materials and Methods.—Two radiation-resistant biochemical mutants of strain B of *E. coli* were chosen for use in these studies because of the ease with which back mutations of these organisms could be screened and assayed. The first is a streptomycin-dependent mutant[11] which grows well at streptomycin concentrations of between 1 and 100 μg. per ml. This organism was cultured and assayed for survival and back mutations by methods developed by Bertani.[12] Stock cultures were maintained on nutrient agar slants containing 25 μg. of streptomycin per ml. Cells used for mutation studies were grown for 18 hrs. with aeration at 37°C. in nutrient broth containing 10 μg. of streptomycin per ml. Survival assays were made by surface plating, from appropriate dilutions, on nutrient agar containing 25 μg. of streptomycin per ml. Assays for the back mutation to streptomycin non-dependence were made by plating on nutrient agar without streptomycin. The second organism is a purineless mutant[13] whose growth factor requirement is satisfied by adenine. This organism is best maintained on a rich nutrient agar. Cells used for mutation studies were grown for 15 hrs. with aeration at 37°C. in nutrient broth. Survival assays were made on nutrient agar while assays for the back mutation to purine non-dependence were made on a purine-free synthetic medium containing Noble agar, phosphate buffer, ammonium salts, trace elements, asparagine, glutamic acid, glycine and dextrose. In all cases, assay determinations were based on the average of four aliquot plates. Survival assay plates were incubated 48 hrs. at 37°C. Mutant assay plates were incubated at the same temperature for 120 hrs. due to the slower growth rate of the mutant colonies.

The techniques and apparatus employed for x-ray exposure of cells under high and low oxygen tension were developed by G. E. Stapleton of this laboratory and are described in detail by Hollaender and Stapleton.[14] Only those portions of the technique pertinent to this investigation are reported here. Cells used in all experiments were prepared by washing twice in M/15 phosphate buffer at pH 6.9 with a final resuspension in the buffer to give 2.0–3.5 $\times$ 10^9 cells per ml. The cell suspensions were irradiated in 1-ml. amounts contained in 2-ml. thin-walled volumetric tubes. These tubes were fitted with ground glass stoppers which carried bubbling and exhaust tubes equipped with stopcocks. This arrangement permitted the removal of air from the suspension and the saturation of the suspension and the atmposhere in the tube with the desired gas at a slight positive pres-

sure. In all cases the suspensions were bubbled for 15 or 20 minutes, with the tubes immersed in ice water at 2°C., just prior to irradiation. Control and experimental tubes were maintained at 2°C. until assayed. Pre- and postirradiation treatments by themselves had no effect on the survival and mutation rate of either strain.

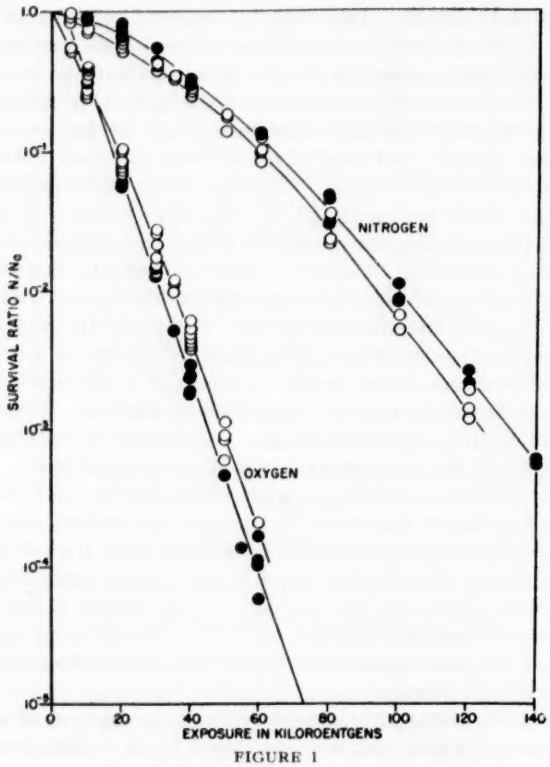

FIGURE 1

X-ray survival curves of the streptomycin-dependent strain (O) and of the purineless strain (●) irradiated in oxygen or nitrogen.

During irradiation the tubes were held at a slight angle in radial holes in a lucite ring-shaped holder. The suspension in each tube was held in the center well of the holder at an equal distance from the center of the vertical beam of x-rays. Thus all tubes were exposed in a predetermined uniform field of radiation. An additional opening in the holder permitted the insertion of a 250-r thimble ionization chamber of a standardized Vic-

toreen dosimeter so that the exposure rate in the exact area containing the suspensions could be measured. The center well of the holder was filled with a water-ice mixture in which the tubes were immersed during irradiation.

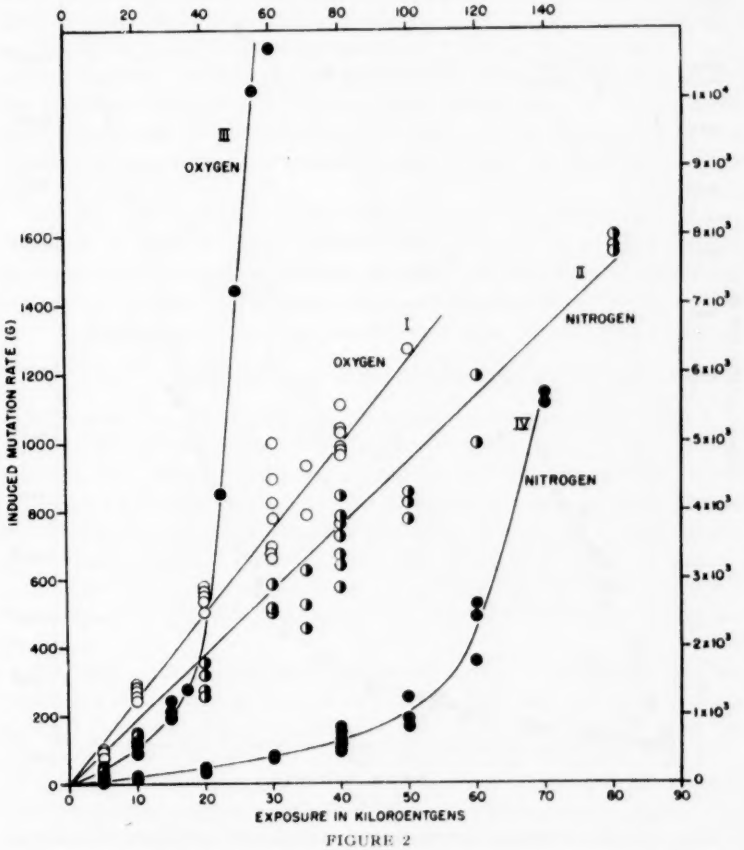

FIGURE 2

X-ray induction of back mutations as a function of kiloroentgens of exposure. Comparison of back-mutation rates to streptomycin non-dependence of the streptomycin-dependent strain when irradiated in oxygen, curve I or in nitrogen, curve II (left and bottom scales). Right and top scales compare the back-mutation rates of the purine-less strain to purine non-dependence when irradiated in oxygen, curve III, with those obtained when irradiated in nitrogen, curve IV.

A General Electric, Maximar Model, 250-kvp. unit operated at 250 kvp. and 15 ma. served as the x-ray source. The inherent filtration was equiva-

lent to 3 mm. of aluminum. All exposures were carried out at a constant rate of 40 kr. per hour at a target distance of 25 cm.

Experimental Results.—Survival ratios, obtained in oxygen and in nitro-

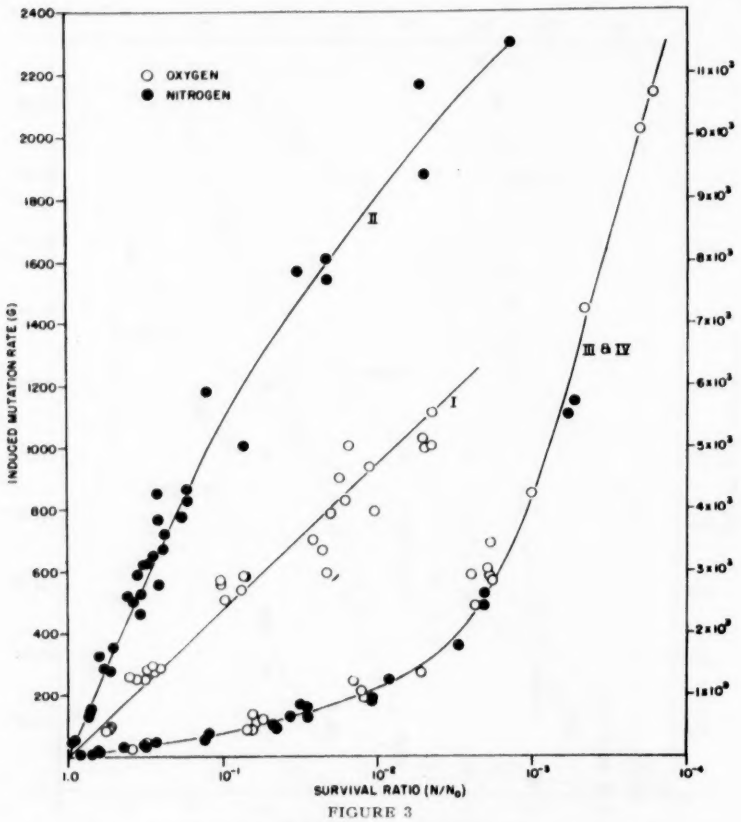

FIGURE 3

Rate of x-ray induction of back mutations as a function of survival ratio. Curves I and II, left scale, compare the back-mutation rates to streptomycin non-dependence of the streptomycin-dependent strain irradiated in oxygen or nitrogen. Curves III and IV, right scale, compare the back-mutation rates to purine non-dependence of the purineless strain irradiated in oxygen or in nitrogen.

gen, plotted against exposure in kiloroentgens are shown in figure 1. Similar, though perhaps not identical, survival curves are obtained for the two strains. Each organism exhibits a much greater sensitivity to x-rays when exposed in oxygen than in nitrogen. At an exposure of 60 kr. a ratio of

survivors in nitrogen to survivors in oxygen of 1300 is shown for the purineless strain and a ratio of 660 for the streptomycin-dependent strain. At a survival ratio of 0.5 per cent a dose-reduction value[15] of about 2.5 is obtained for the streptomycin-dependent strain and about 3.0 for the purineless strain. The survival data indicate that a mechanism which is accelerated by the presence of oxygen is involved in a similar manner in the lethal action of x-rays on these two strains of coli. If this same mechanism acts on the hereditary material of the cells to induce mutations one would expect the mutation rate of cells exposed to x-rays in the presence of oxygen to be greatly increased over that obtained on exposure in nitrogen. This was found to be the case for the purineless strain but not for the streptomycin-dependent strain.

Mutation rates for both strains plotted against exposure in kiloroentgens are shown in figure 2 and against survival ratios in figure 3. The induced mutation rate G is expressed as the number of mutations per 10^8 surviving cells and is corrected for spontaneous mutations present in the suspension prior to irradiation. The term G is obtained from the following equation: $G = \left(\frac{M}{N} - \frac{M_0}{N_0}\right) \times 10^8$, where M is the number of mutations per ml. in the irradiated suspension, M_0 the number of spontaneous mutations per ml. in the unirradiated suspension, N the number of survivors per ml. in the irradiated suspension and N_0 the original number of viable cells per ml.

Curves I and II in figure 2 show that the x-ray-induced mutation rates for the streptomycin-dependent organism are very similar at equal exposures whether the cells are irradiated in the presence of oxygen or of nitrogen. This is in sharp contrast to the very great difference in survival levels with respect to exposure obtained with oxygen and nitrogen for this organism.

Mutation rates plotted against survival ratios for the streptomycin-dependent strain, curves I and II in figure 3, show that at equal levels of survival in the two gases the mutation rate in nitrogen is 2–2.5 times the value obtained in oxygen. The cells irradiated in nitrogen received approximately 2.5 times the x-ray exposure received by the cells at an equal survival level in oxygen, as shown in figure 1. Therefore the results presented for the streptomycin-dependent strain in figures 2 and 3 are such as might be expected on the basis of a direct action of x-rays on the hereditary components of the cells responsible for the back mutation to streptomycin non-dependence. Although oxygen has a pronounced influence on the lethal effects of x-rays for the streptomycin-dependent strain it has but slight influence on the genetic effect examined. The mechanisms involved in producing the lethal effect of x-rays on this strain of coli and the mutagenic effect of x-rays for this particular mutation appear to be quite different.

The back mutation rates of the purineless strain plotted against kiloroentgens of exposure are shown in figure 2, curves III and IV. It is obvious that the results obtained with this strain are in sharp contrast to those obtained with the streptomycin-dependent strain. The x-ray-induced back mutation rates to purine non-dependence are profoundly influenced by a high oxygen tension during irradiation. This is clearly shown by a comparison of the mutation rates, with respect to x-ray exposure, obtained in

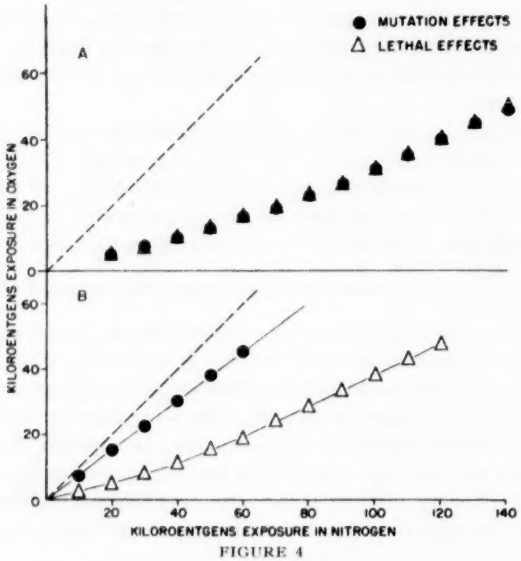

FIGURE 4

Isoefficiency curves. The abscissa represents the kiloroentgens of exposure in the presence of nitrogen required to produce the same lethal or genetic effect as the exposure in kiloroentgens in the presence of oxygen plotted on the ordinate, (*A*) for the purineless strain, (*B*) for the streptomycin-dependent strain. In each case the dotted line represents the curve that could be expected if both gases exerted an equal or no influence on the effects examined. Points lying below this curve indicate an increased effect of oxygen over that of nitrogen.

oxygen and in nitrogen. In the presence of oxygen an exposure of 50 kr. induced a mutation rate of 7200 mutations per 10^8 surviving cells. The same exposure in the presence of nitrogen resulted in a mutation rate of only 300 mutations per 10^8 survivors. The dose-reduction value of oxygen for the genetic effect studied is approximately 3 (curves III and IV in figure 2). This agrees closely with the dose-reduction value obtained for the lethal effect with this strain (figure 1).

The similarity of the dose-reduction values for the two effects studied indicates that oxygen plays essentially the same role in increasing both the killing and the mutagenic effect of x-rays in the case of the purineless strain of coli. This is corroborated by the data presented in figure 3, curves III and IV. From this it may be seen that the mutation rates plotted against survival rates for cells x-rayed in the presence of oxygen and of nitrogen give curves which coincide. Therefore the induced back-mutation rate for this particular genetic effect is the same at equal survival levels irrespective of whether the cells have been exposed to x-rays in the presence or the absence of oxygen. The cells at any survival level in oxygen, however, received approximately one-third the amount of energy received by the cells in nitrogen at an equal level of survival.

Discussion.—The influence of oxygen on the x-ray-induced mutation rates of the two strains examined represent two extreme conditions that could be expected. This is shown in a more obvious manner in figures 4 (*A*) and 4 (*B*) in which portions of the data presented in figures 1 and 2 have been replotted. In these two graphs the x-ray exposure necessary to produce a certain degree of effect in the presence of oxygen is plotted as a function of the exposure in the presence of nitrogen required to produce the same degree of effect.

Figure 4 (*A*) represents the results obtained with the purineless strain. The curves for the lethal and the genetic effects coincide. Oxygen has the same efficiency in increasing the x-ray induction of the back mutation to purine non-dependence as it has in increasing the lethal effects of x-rays.

This relationship is consistent with the idea that x-rays produce an active molecule species or radical which is responsible for both the genetic and lethal effects in the purineless strain. The rate at which this substance is produced or activated, and hence the degree of genetic and lethal effects obtained, is dependent on the presence of oxygen during the irradiation of the cells.

Figure 4 (*B*) presents the opposite picture. The idea that a single substance produced by x-rays may be responsible for both the lethal and genetic effects in the streptomycin-dependent strain is not compatible with the results observed. Although oxygen has nearly the same degree of efficiency in increasing the lethal effects of x-rays on both strains, it has but slight influence on the back-mutation rate to streptomycin non-dependence. This difference in response to the presence of oxygen suggests that the mechanisms responsible for the lethal and this specific genetic effect of x-rays are different in this strain. The genetic effect may be the result of the action of an oxygen-insensitive substance produced by the radiation, or of a direct action of x-rays. The small oxygen effect observed on the back-mutation rate to streptomycin non-dependence may be real or apparent. One possible explanation is that more than one type of back mutation to

streptomycin non-dependence, with a constant ratio between them, may occur. The mechanism involved in inducing the major type of mutation should be insensitive to oxygen while that for the less frequent types would be oxygen sensitive. The assumption that oxygen exerts a slightly protective action on the newly formed mutants, rendering them less sensitive than the normal cells to the lethal effects of x-rays, could also possibly explain the slight oxygen effect.

The mutation rates of the streptomycin-dependent strain as a function of exposure in kiloroentgens give curves which are approximately first order (curves I and II in figure 2). This may represent a single-hit type of phenomenon and thus indicate that a back mutation in the streptomycin dependent strain is the result of a single ionization.

The mutation rates of the purineless strain in the presence of oxygen, plotted as a function of the exposure in kiloroentgens, give curves which are approximately fourth order (curve III, figure 2). This may be interpreted as an indication that a back mutation in the purineless strain does not result from a single ionization or from the action of a single individual molecule or radical but may require the cooperative action of several of the same species.

On the basis of the two genetic effects examined it may be postulated that different mechanisms may be involved in the x-ray-induced mutation of different genes. Further studies with other micro-organisms and a more significant number of mutations may show this technique to be a powerful tool in differentiating mutations.

These studies are being continued and will be presented in greater detail in another publication.

Summary.—The effect of a high oxygen tension on the induction of mutations by x-rays has been examined for a streptomycin-dependent strain and for a purineless strain of *E. coli*. Back-mutation rates to streptomycin non-dependence and purine non-dependence were studied under comparative exposures to x-rays in the presence of oxygen and of nitrogen. The sensitivity of both strains to the lethal effects of x-rays was similarily increased by the presence of oxygen during irradiation. A dose-reduction value of 2.5 was found for the streptomycin-dependent strain and a value of 3 found for the purineless strain. Mutation rates of both strains were increased with length of exposure to x-rays in the presence of either oxygen or nitrogen. Oxygen was found to have but slight additional effect over nitrogen on x-ray induction of back mutations in the streptomycin-dependent strain. On the other hand the back-mutation rate of the purine-dependent strain was found to be profoundly increased by the presence of oxygen during irradiation. The dose-reduction value of oxygen for the genetic effect for this strain is the same as that for the lethal effect. This is interpreted as an indication that the same mechanisms are involved in the lethal and

this specific genetic effect of x-rays in the purineless strain. Different mechanisms appear to be involved in these two effects of x-rays on the streptomycin-dependent strain.

Acknowledgment.—Grateful acknowledgment is made to Mrs. Ruth W. Whittle for technical assistance in these experiments and to Mr. George E. Stapleton for his helpful advice on the techniques used in exposing cells under high and low oxygen tension.

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[15] The term dose-reduction as used in this paper is the ratio of the exposure in nitrogen to that in oxygen which produces the same degree of effect. See Kelner, A., *J. Bact.*, **58,** 511 (1949).

GENE-CONTROLLED TIME CONSTANTS IN CONVULSIVE BEHAVIOR

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Communicated by C. C. Little, April 7, 1951

Certain strains of mice have been known for several years to differ in susceptibility to sound-induced convulsions.[1] In a previous publication[2] the period of time elapsing between the onset of the sound stimulus and the beginning of the convulsion (latency) was shown to be a genetic character. The DBA/2 strain, with 99 per cent convulsion risk at 30 days of age, had an average latency of 35.8 seconds, while its hybrid with the C57

black strain had at the same age a convulsion risk of 72 per cent and an average latency of 55.2 seconds. Fifty C57 blacks used in this experiment gave no convulsions in five trials each. It was further found that within a genetically homogeneous group there was no relationship between latency of a convulsion and the probability that an animal would die or recover as a result of a convulsion.

Three independent hypotheses were formulated to explain the results of this experiment. The first part of this paper is concerned with experiments designed to test each hypothesis by using a third strain of inbred mice (A Heston), and studying the effects of hybridization between this strain and the two used in the original study (C57 Bl and DBA/2). Cousin matings were substituted for brother-sister matings for one generation at the time the authors established the A stock, but this is not believed to have significantly affected its isogenicity. Tests were made in the manner described

TABLE 1

CHARACTERISTICS OF SOUND-INDUCED CONVULSIONS IN THREE INBRED STRAINS OF MICE AND THEIR HYBRIDS AT 30–40 DAYS OF AGE

MATING	TRIALS	CONVULSION RISK, %	MEAN LATENCY, SECONDS	S. D. OF LATENCY	ADJUSTED MEAN LATENCY	DEATH RISK OF CONVULSER
DBA × DBA	149	92	35.8	12.5	38.3	87
DBA × A	97	91	35.6	16.6	39.4	21
DBA × C57 Bl	156	76	52.1	9.7	56.5	59
A × A	144	11	61.8	12.2	75.7	0
A × C57 Bl	156	4.5	54.6	*	74.6	57
C57 Bl	250	0	..	..	..	..

* Too few convulsions to calculate a standard deviation. A value of 12.5 was assumed for determining the adjusted mean latency.

previously,[2] except that forty-eight hours always intervened between trials. This period has proved sufficient for the disappearance of any effects of a previous test on susceptibility.

The Threshold Hypothesis.—A particular convulsion risk is dependent upon the number of susceptibility producing genes with additive effects present in the animal. Each genotype determines the range of positions of an animal on a continuous scale of susceptibility, which is divided by a threshold into convulsing and non-convulsing portions. The DBA/2's are almost always above threshold; the C57 blacks are almost always below. Their F_1 hybrid is above threshold 72 per cent of the time. The A strain was found to have a convulsion risk of 11 per cent. According to the hypothesis stated above, A × DBA/2 hybrids should have a convulsion risk above 72 per cent, and A × C57 black hybrids should have a risk below 11 per cent. These predictions were confirmed by the experimental results. (Table 1, column 3.)

The Accommodation Hypothesis.—The occurrence or non-occurrence of a convulsion under auditory stimulation depends upon the outcome of a race between an excitatory process set up by the sound, and a gene-controlled process of accommodation occurring within the organism. Animals with a slow rate of adaptation will have a high convulsion risk, and these convulsions will occur after a short latency. Animals with a rapid rate of accommodation will not convulse at all, while strains with intermediate rates of adaptation will contain some members in whom accommodation wins out, and others in whom convulsions will occur. The latencies of convulsions in such a strain will tend to be longer than those of strains with a high convulsion risk.

If this view is correct, placing the strains in the order of convulsion risk will also arrange them in the order of convulsion latencies. This prediction is verified by the results with the A strain and its hybrids, as shown in column 4, table 1. Only the A × C57 black hybrids are slightly out of line, and the number of convulsions in this group is so small that sampling errors may be large. The observed latencies may not be the most satisfactory way to compare the relative rates of accommodation within a group of mice. The mean latency is based upon only those animals which convulse; that is, upon the most susceptible animals in the group. If latency is a function of the rate of accommodation, and this character is normally distributed within the population, it is possible to calculate a presumptive latency for the non-convulsing animal by means of the inverse probability[3] transformation. These values are given in column 6 of table 1. Since the number of convulsing animals in the A × C57 black hybrids was too low to calculate a standard deviation, a standard deviation of 12.5 seconds, which is close to the average of other groups, was assumed. These results support the view that the underlying variable determining seizure susceptibility is the rate of accommodation which may be above or below a critical threshold.

The Independence of Susceptibility and Outcome.—The third hypothesis, which was tested with the A strain and its hybrids, holds that the risk of death following a convulsion is determined by a different set of genes and different physiological factors than those which determine convulsion risk. Column 7 of table 1 shows that death risk bears no necessary relationship to convulsion risk. The A × DBA/2 hybrids have a very high convulsion risk and a relatively low death risk. The evidence indicates that C57 blacks would not be particularly resistant to death if they were susceptible to convulsions, since their hybrids do not show the relatively great ability to recover from convulsions which is shown by A's and by A hybrids.

Kinetics of Accommodation.—The second set of experiments was designed to investigate the kinetics of the excitatory and adaptive processes in seizure susceptible mice. These experiments involved a preliminary priming

stimulation, an interval of no stimulation and a prolonged test stimulation. Since convulsions occur rarely after 60 seconds of stimulation, and practically never after 90 seconds, the latter duration was selected for the test period.

With each temporal arrangement, observations were made on the occurrence of seizures and on the total stimulus time (TST) required to produce a convulsion. If the TST necessary to produce convulsions decreases, it may be reasoned that the priming stimulus has a certain momentum which carries over across the interval. If the TST remains the same it may be concluded that the priming stimulus produces an excitatory state which persists unchanged over a certain period of time.

Finally, if the necessary TST increases, or if the animal actually becomes refractory to stimulation, it may be assumed that the interruption has somehow produced a decreased excitability of the motor centers. The duration of this refractory period may be studied by lengthening the interval between priming and test simulation until susceptibility to convulsions again returns.

Subjects of this experiment were 30- to 35-day-old hybrid mice from crosses between the DBA/2 and the C57 Black/6 strain. The apparatus for stimulation was modified from that previously described[2] by suspending a doorbell across the top of the wash tub used for a test enclosure by means of rubber bands, instead of hanging the bell over the side. Intensity of the sound measured by an H. H. Scott meter was approximately 91 db. above 0.0002 dyne per cm.^{2} R. M. S. pressure. A control group of animals was selected at random from a large number of litters, and any one litter was used for several experimental groups in order to avoid loading any group with a number of individuals which might be in some way abnormal. Control animals were tested with 90 seconds of continuous bell ringing.

The procedure for each test in the summation series included a priming stimulation of either 5, 10 or 20 seconds, an interrruption of from 5 to 40 seconds, and continuous stimulation for 90 seconds or until the animal convulsed. The record of each trial included the behavior of the subject during each phase, the time of onset and type of convulsion if one occurred, and the outcome of the convulsion. Mice which would not convulse or which survived a convulsion were used for one other test after an interval of forty-eight or more hours.

Behavioral Observations.—The behavior of mice during the three phases of the trial is remarkably constant. The priming stimulus generally brings forth a startle and a short period of wild running after 5 or 10 seconds of stimulation. In the DBA/2's this results in a full-fledged convulsion in approximately 25 per cent of the cases. Such individuals could not, of course, be used for the interrupted stimulus experiments. The early wild running in the hybrids practically never develops into a convulsion. When

it does not terminate in a convulsion, wild running stops spontaneously and the animal sits tensely for a period, usually quivering and often with the tail pointing directly up. During the interruption every animal sat still for periods of 15 to 20 seconds. If the interval was longer than this the subjects might walk around but never showed signs of preconvulsive activity. When the stimulus was resumed, after the shorter intervals, there was often a rapid burst of activity which terminated in a convulsion. It appeared in some cases that the animal was just

TABLE 2

ABBREVIATED SUMMARY OF EXPERIMENTS ON THE EFFECT OF TEMPORAL STIMULUS PATTERN ON AUDIOGENIC SEIZURES IN MICE. RESULTS WHICH DIFFER SIGNIFICANTLY FROM THE CONTROL ARE FOLLOWED IN PARENTHESES BY THE PROBABILITY THAT THE RESULT COULD OCCUR BY CHANCE

GROUP	N	MEAN TST SEC.	TST LESS THAN 30 SEC.		CONVULSIONS		DEATHS	
			OBSERVED	EXPECTED	OBSERVED	EXPECTED	OBSERVED	EXPECTED
Control	40	48.5	0	0	34	34	20	20
All 5-sec. priming groups	70	47.5	11 ($p < 0.001$)	0	64	59.5	29 ($p < 0.05$)	38
All 10-sec. priming groups	105	37.0 ($p < 0.01$)	39 ($p < 0.001$)	0	89	89.3	41 ($p < 0.02$)	52
20-sec. priming; 5-, 10-, 15-sec. int.	45	53.5 ($p < 0.01$)	1	0	36	38.3	16	21
20-sec. priming; 20-, 25-, 30-sec. int.	45	71.0 ($p < 0.001$)	0	0	15 ($p < 0.001$)	38.3	2 ($p < 0.001$)	9
20-sec. priming, 40-sec. int.	15	.....	0	0	0 ($p < 0.001$)	12.7	.....	..
20-sec. priming, 1-min. int.	15	59.0	0	0	1 ($p < 0.001$)	12.7	0	0.6
20-sec. priming; 2-min. int.	15	47.6	0	0	8	12.7	2	4.7
20-sec. priming; 5-min. int.	15	55.7	0	0	10	12.7	2	5.9
20-sec. priming; 10-min. int.	15	63.7 ($p < 0.01$)	0	0	10	12.7	3	5.9
Sum of three lower rows	45	.....	0	0	28 ($p < 0.001$)	38.1	7 ($p < 0.001$)	16.5

on the verge of convulsion and merely needed a slight push to bring on the grand mal discharge. Certain combinations of priming stimulus and interval produced a lowered excitability, and it will be seen from the results that it is possible to produce complete refractoriness by proper manipulation of the temporal pattern of stimulation. Refractory animals show no sign of preconvulsive behavior and very little activity except walking or grooming during the test stimulation period.

Results of Interrupted Stimulation.—Table 2 gives the results of these experiments in condensed form. Significant differences between the experimental and control groups appear in total stimulus time, the number of individuals with TST's of less than 30 seconds, the susceptibility to convulsion and the outcome of convulsions. In this table, values of p are given in parentheses, where p is the probability that the difference between observed and expected results could occur by chance sampling. The expected numbers of convulsers and deaths were calculated from the convulsion risk and death risk of the control sample.

All of the 5-second priming groups, 70 trials in all, may be considered together since there appears to be little difference between the effects of a 5-second and a 40-second interruption. There is a slight and possibly significant ($P < 0.10$) increase in seizure risk over the control. The mean TST is almost identical with the control value, but there is a highly significant increase in the number of animals convulsing with less than 30 seconds of stimulation. The mean TST does not change because those animals which do not convulse rapidly are slightly retarded. In about one-sixth of the cases the priming excitation carries across the interruption and the test stimulus brings on an accelerated convulsion. In the other five-sixths of the animals the priming excitation is ineffective and the whole process must be started anew. In these animsls the TST is increased about 5 seconds. The death risk of convulsers is significantly reduced.

Similarly it has been possible to group together 105 ten-second priming tests, since the length of the interruption, at least up to 40 seconds, does not appear to modify the results. This pattern of stimulation has no effect upon the convulsion risk, but four-ninths of the animals gave accelerated convulsions. The remaining animals were retarded by an average of 10 seconds. The net results of these two effects are to decrease the mean TST of these groups by 11.5 seconds. A marked reduction in death risk is found in this group also. In general, 5-second priming and 10-second priming periods have similar effects but the excitation developed in a 10-second period has a greater tendency to carry over.

Twenty-second priming has a markedly different effect which varies with the length of the interruption period. A fairly sharp break seems to exist between the results of interruptions of 15 seconds or less and those of 20 seconds or more. Neither group has an appreciable number of accelerated

convulsers, and the mean TST is significantly increased in both. The most striking finding is the fall in seizure susceptibility following interruptions of 20 seconds or more. A schedule of 20 seconds of priming and 40 seconds of interruption appears to give almost total immunity. The death risk is further reduced in the 20-second priming groups.

It appears that some critical change in the nervous system occurs in the period between 10 and 20 seconds of auditory stimulation. Before this point is reached a priming stimulus is either neutral or enhances convulsion risk. After this point an interruption permits a decline in excitability which eventually results in refractoriness comparable to that which follows an actual convulsion. Its physiological basis is unknown. It may be a change in the intrinsic characteristics of the nervous system itself, or it may depend upon indirect effects, such as secretion of hormones or cardiovascular phenomena. The duration of the refractory period was studied by extending the length of the interruption following 20-second priming periods. The results show a sharply increasing susceptibility between 1 and 2 minutes with almost normal susceptibility after 2 minutes, though death risk is significantly reduced and TST is increased.

Discussion.—The time course of events in audiogenic convulsions of DBA/2 × C57 black hybrids may be deduced from the data presented. Sound stimulation above a certain level sets up a relatively persistent excitatory focus within 5 to 10 seconds. Since the duration of the interval following 5- or 10-second priming stimulation seems unimportant, individual differences must be related to the time required to establish the excitatory focus. Persistent stimulation for 20 seconds brings this focus to a critical point. If stimulation is continued, or if it is interrupted for not more than 15 seconds, a generalized convulsion may occur. Longer interruptions permit the excitation to discharge and result in a period of inexcitability which persists between 1 and 2 minutes. In a certain percentage of cases the convulsion threshold is not reached, even with continuous stimulation, and an inhibitory state is produced after about 60 seconds.

The time course of these events is comparable, though slightly longer, than the times involved in the experiments of Dusser de Barenne and McCulloch[4] on faradic stimulation of the cortex. The important difference in this case is that excitation arises from stimulation of an afferent nerve by its natural receptor organ. The relative constancy and predictability of a genetically controlled population makes the behavior of the whole organism amenable to techniques similar to those used by the Oxford School in their studies of spinal reflexes.[5] Since the kinetics of the convulsive process is gene-controlled, the various inbred strains of mice must differ quantitatively in some feature which affects excitability of the cortex.

A hypothesis which can be tested experimentally and which is consistent with theories of gene action and of cerebral physiology, assigns this dif-

ference to enzyme systems which control metabolic processes affecting cerebral pH. Such differences may be localized in the nervous system, or may reside elsewhere.

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[2] Fuller, J. L., Easler, C., and Smith, M. E., *Genetics*, **35**, 622–632 (1950).
[3] Wright, S., *J. Am. Statis. Ass.*, **21**, 162–178 (1926).
[4] Dusser de Barenne, J. G., and McCulloch, W. S., *J. Neurophysiol.*, **2**, 319–355 (1939).
[5] Creed, R. S., Denny-Brown, D., Eccles, J. C., Liddell, E. G. T., and Sherrington, C. S., *Reflex Activity of the Spinal Cord*, Oxford, 1932, 182 pp.

COMPLEX SPACES WITH TRANSITIVE COMMUTATIVE GROUPS OF TRANSFORMATIONS

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In algebraic geometry there is a theorem known to the effect that if on an algebraic variety there is given a transitive commutative Lie group of birational transformations then the variety can be uniformized by hyperelliptic functions of the same dimension as the variety,[1] and as a comment on this theorem we will give a proposition which is rather more comprehensive than itself.

Theorem. *Let V_{2k}, $k \geq 1$, be a (non-algebraic, non-compact) complex coordinate space of $2k$ real dimensions. If, for $r \geq k$, there are on it r holomorphic contravariant vector fields*

$$\eta_p{}^{\alpha} \qquad \alpha = 1, \ldots, k; \quad p = 1, \ldots, r, \tag{1}$$

such that the rank of the matrix (1) has everywhere its maximum value k ("transitivity") and that the bracket expressions

$$\varphi_{pq}{}^{\alpha} \equiv \eta_p{}^{\beta} \frac{\partial \eta_q{}^{\alpha}}{\partial z^{\beta}} - \eta_q{}^{\beta} \frac{\partial \eta_p{}^{\alpha}}{\partial z^{\beta}}$$

all vanish identically

$$\varphi_{pq}{}^{\alpha} = 0, \tag{2}$$

("commutativity"), then there are on the space k simple Abelian integrals of the first kind by which it is mapped holomorphically and locally one-one into the Euclidean E_{2k}. In particular,[2] if V_{2k} is compact it is a complex multitorus.

Proof: As on a previous occasion[3] we introduce the Hermitian tensorfield

$$g^{\alpha\beta^*} = \sum_{p=1}^{r} \eta_p{}^{\alpha}\overline{\eta_p{}^{\beta}}.$$

Due to the transitivity postulated, it is strictly positive definite and thus has an inverse $g_{\alpha\beta^*}$, and for the latter we claim the Kaehler property

$$\frac{\partial g_{\alpha\beta^*}}{\partial z_\gamma} - \frac{\partial g_{\gamma\beta^*}}{\partial z_\alpha} = 0. \tag{3}$$

In fact,[3] equation (3) is equivalent with

$$\frac{\partial g^{\alpha\rho^*}}{\partial z_\beta} g^{\beta\sigma^*} - \frac{\partial g^{\alpha\sigma^*}}{\partial z_\beta} g^{\beta\rho^*} = 0$$

which in turn is equivalent with

$$\sum_{p,q} \overline{\eta_p{}^{\rho}}\,\overline{\eta_q{}^{\sigma}}\,\varphi_{pq}{}^{\alpha} = 0$$

and thus is certainly implied by (2).

We next claim that the given vector fields (1) are self-parallel in the constructed metric, that is

$$\frac{\partial \eta_p{}^{\alpha}}{\partial z^{\beta}} + \eta_p{}^{\gamma}\Gamma_{\gamma\beta}{}^{\alpha} = 0. \tag{4}$$

In fact, due to [4]

$$\Gamma_{\beta\gamma}{}^{\alpha} = -\frac{\partial g^{\alpha\rho^*}}{\partial z_\beta} g_{\rho^*\gamma},$$

equation (4) is equivalent with

$$\frac{\partial \eta_p{}^{\alpha}}{\partial z_\beta} g^{\beta\rho^*} = \eta_p{}^{\gamma} \frac{\partial g^{\alpha\rho^*}}{\partial z_\gamma},$$

and hence with

$$\sum_{\varphi} \overline{\eta_q{}^{\rho}} \varphi_{pq}{}^{\alpha} = 0,$$

and this is again implied by (2). It is actually equivalent with (2) but there is no need for verifying this. Now, this implies that in the Ricci relation

$$\eta^{\alpha}_{p,\ \beta,\ \gamma^*} - \eta^{\alpha}_{p,\ \gamma^*,\ \beta} = \eta_p{}^{\delta} R_{\delta\beta\gamma^*}{}^{\alpha}$$

the left side vanishes identically, and hence also the right side. But (1) has everywhere maximal rank, so that

$$R_{\delta\beta\gamma^*}{}^{\alpha} = 0,$$

and thus our space is a flat Kaehler space. Therefore, in the neighborhood

of every point we can allowably normalize the metric tensor to

$$g_{\alpha\beta^*} = \delta_{\alpha\beta^*} \tag{5}$$

and therefore k linearly independent Abelian integrals of the stated kind can be introduced locally. However, since the normalization (5) can be introduced in every neighborhood, such Abelian integrals can be continued along every path and they remain linearly independent in the neighborhood of every point, which completes the proof of the theorem.

Actually, the normalization (5) shows that the contravariant vector fields

$$\zeta_\alpha{}^r = g_{\alpha\beta^*}\overline{\eta_r{}^\beta}$$

are likewise holomorphic and self-parallel, so that in particular we have

$$\frac{\partial \zeta_\alpha{}^r}{\partial z_\beta} - \frac{\partial \zeta_\beta{}^r}{\partial z_\alpha} = 0.$$

Therefore the r Abelian integrals

$$w^p(z) = \int_{z^0}^{z} \zeta_\alpha{}^p \, dz_\alpha$$

may be introduced, and if, for instance, the first k among them are linearly independent at the point z^0, then they will be so everywhere and offer the mapping claimed.

Remark.—We will now describe a general possibility for constructing a Kaehler metric (not necessarily a flat one) from given data by an old idea of M. Noether which actually only performs a holomorphic immersion into a complex projective space of sufficiently many dimensions and thus induces a Kaehler metric automatically.

Assume at first that we are given a number of scalar densities

$$\varphi_q(z), \qquad q = 1, \ldots, s \tag{6}$$

$s > k + 1$, each being a one-coordinate object which under a change of coordinates transforms itself by a multiplicative factor $\left(\frac{\partial(z)}{\partial(z')}\right)^g$ where g is any integer, positive or negative, the same for all. If then the s by $k + 1$ matrix

$$\left| \varphi_q, \frac{\partial \varphi_q}{\partial z_\alpha} \right| \qquad q = 1, \ldots, s; \quad \alpha = 1, \ldots, k, \tag{7}$$

has everywhere its maximal rank $k + 1$, then the equations

$$w_q = w_q(z)$$

will map our space V_{2k} holomorphically and locally regularly into the projective space P_{2s-2} of the points

$$w_1 : w_2 : \ldots : w_s.$$

Suppose now that we are given a holomorphic tensor density $\zeta^{\alpha_1 \ldots \alpha_m}_{\beta_1 \ldots \beta_n}$ of a given type $(m;\ n;\ g)$, with "symmetries" added. If the number of independent tensor components is N, then we denote this object by one cumulative index. Thus: ζ^A, and we envisage the presence of several such tensor-fields

$$\zeta_p{}^A, \quad A = 1, \ldots, N; \quad p = 1, \ldots, r \tag{8}$$

all of the same type, and with $p \geq N + 1$. Now, it is possible to verify that the N-dimensional square matrices of the matrix (8), whose number is $s = \binom{r}{N}$, are all densities of a certain weight; and if we denote them by (6) and impose the previous condition on the matrix (7) then a mapping into a projective space will ensue. The condition on the matrix (7) could probably be stated more simply in terms of the elements (6) itself, but we will not pursue this point any further.

[1] Due to Picard, Painlevé and others. For references see Castelnuovo, G., and Enriques, F., "Die Algebraischen Flächen, etc." *Encyclopädie der Mathematischen Wissenschaften*, Vol. III 21, Article III C 6b, pp. 744–746.

[2] Compare our "Vector Fields on Complex and Real Manifolds," *Ann. Math.*, **52**, 645–646 (1950).

[3] "Vectorfields and Ricci Curvature," *Bull. Am. Math. Soc.*, **52**, 796–797 (1946).

[4] "Curvature in Hermitian Metric," *Ibid.*, **53**, 180 (1947).

ON THE EXISTENCE OF A BASIS FOR EVERY FINITE ABELIAN GROUP

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This note presents a simple constructive type of proof of the theorem indicated in the title. The proofs in the standard treatises (e.g., Weber, H., *Lehrbuch der Algebra*, **2**, 42–45 (1899)) are fairly complicated, usually involving an inductive process.

Another proof, based on a certain minimal condition, will be published in a subsequent issue of these PROCEEDINGS.

If an arbitrary finite set of n-component vectors are arranged in *any* order, and we strike out successively every vector expressible as a linear combination of other vectors as yet unstricken, then the vectors remaining at the end of the process constitute a *basis* for the given set—the proof is almost banal.

On the other hand, if a similar procedure is applied to a finite abelian group G—i.e., if we arrange the elements of G in an *arbitrary* order and then strike out successively each element expressible as a product of powers of other elements as yet unstricken, then generally what remains at the end of the process will *not* be a basis for G.

Under the supposition that G is of prime-power order, the point of the present note is that if we begin the process described above by arranging the elements of G in any *non-ascending* order of their *periods*, then we do obtain as end result a basis for G. The general case follows from the fact that any abelian group can be expressed as direct product of abelian groups of prime-power order. (Cf. Weber, *loc. cit.*, pp. 40–42.)

1. By a *basis* of G we understand a complete and independent set S of elements $\alpha, \beta, \ldots, \lambda$ of G. *Complete* means that an arbitrary element θ of G has a representation of the form

$$\theta = \alpha^a \beta^b \ldots \lambda^l, \tag{1}$$

where $a, b, \ldots, l$ are integers. *Independent* means that no relation of the form

$$\beta^b \epsilon^e \ldots \kappa^k = 1 \tag{2}$$

subsists, where $\beta, \epsilon, \ldots, \kappa$ are a selection of elements of S and the exponents are positive integers each less than the period of the corresponding element: $0 < b <$ period of β, etc.

It is evident, then, that the characteristic property of a basis S of G is that each element θ of G has one and only one representation in the form (1)—two representations, (1) and

$$\theta = \alpha^{a'} \beta^{b'} \ldots \lambda^{l'}, \tag{1'}$$

being considered the same if and only if

$$a' \equiv a, \quad b' \equiv b, \ldots, \quad l' \equiv l,$$

each congruence being modulo the period of the corresponding element.

Suppose the order of G to be $N = p^n$, p a prime. Then the period of each element A_i of G is a power of p, say p^{m_i}.

Let the elements of G be so arranged:

$$G = A_1 A_2 \ldots A_i \ldots A_N, \tag{3}$$

that $i < j$ implies $m_i \leqq m_j$—i.e., in a non-ascending order of the periods.

The leading element A_1 can certainly be generated by the other elements of G (if we omit the trivial case of a cyclic group of order 2). Otherwise these $N - 1$ elements would form a subgroup of G; but if $N > 2$, $N - 1$ does not divide N.

We place a bar over A_1. Then we move systematically to the right in

G, placing a bar over each element that can be generated by the *as yet* unbarred elements distinct from itself, and leaving unbarred any elements that cannot be so generated.

Our theorem is: *the unbarred elements form a basis of* G.

Certainly unbarred elements will be met with—at A_a, last element such that $m_a = m_1$, if not before. For in case all the elements preceding A_a are barred, then the only elements allowed in the attempt to generate A_a are those of a lower period. But these elements θ form a group by themselves, as is seen from the fact that they are characterized by the condition $\theta^q = 1$ where $q = p^{m_a - 1}$. Therefore the elements θ can never generate A_a, which lies outside their group.

2. Let us separate the barred and unbarred elements, being careful to preserve the relative position of the elements of each kind. We write

$$S = A_1 A_2 A_3 \ldots \tag{4}$$

for the unbarred, and

$$T = \bar{A}_1 \bar{A}_2 \bar{A}_3 \ldots 1 \tag{5}$$

for the barred. Here the elements have been renumbered so that the subscript indicates position among elements of its own kind. Thus A_1 in S is not the same as A_1 in G.

3. We have first to prove that S is a *complete* set of generators for G.

Suppose that this is not the case. Then in moving through the series T from *right to left*, we shall come to a first element $\bar{A}_k$ which cannot be generated by S.

However, $\bar{A}_k$ *can* be generated by S together with the elements of T to the right of $\bar{A}_k$, for these were the unbarred elements at the time we came to $\bar{A}_k$ as element of G. But the elements of T to the right of $\bar{A}_k$ can be generated by S, since, by assumption, $\bar{A}_k$ is the *first* element of T counting from the right which cannot be generated by S. Hence $\bar{A}_k$ *can* be generated by S.

This contradiction proves the completeness of S.

4. Suppose, with the purpose of reducing this assumption to an absurdity, that the elements of S are not independent, e.g.—with sufficient typicality—

$$A_1^{h_1} A_3^{h_3} A_6^{h_6} A_7^{h_7} = 1 \tag{6}$$

where

$$0 < h_1 < p^{m_1}, \ \ldots, \ 0 < h_7 < p^{m_7}. \tag{7}$$

Let p^r denote the highest power of p that divides h_1, h_3, h_6, h_7; then

$$(A_1^{k_1} A_3^{k_3} A_6^{k_6} A_7^{k_7})^{p^r} = 1 \tag{8}$$

and at least one of k_1, k_3, k_6, k_7 is not divisible by p—say k_6. We write

$$A_1^{k_1}A_3^{k_3}A_6^{k_6}A_7^{k_7} = E, \tag{9}$$

and have $E^{p^r} = 1$, so that the period of E is p^s where $s \leqq r$.

Since p does not divide k_6, we have the existence of two integers x, y such that

$$xk_6 + yp^{m_6} = 1. \tag{10}$$

Raise (9) to the x power, and

$$A_6^{p^{m_6}} = 1 \tag{11}$$

to the y power, then multiply; we find, with attention to (10),

$$A_1^{k_1x}A_3^{k_3x}A_6A_7^{k_7x} = E^x; \tag{12}$$

therefore

$$A_6 = A_1^{-k_1x}A_3^{-k_3x}A_7^{-k_7x}E^x. \tag{13}$$

This expresses A_6 as a power-product of other unbarred A's together with an element E whose period $\leqq p^r$. But p^r divides h_6, which by (7) is less than p^{m_6}; therefore $p^r < p^{m_6}$. Hence the period of E is less than that of A_6.

Consequently, also E, as well as A_1, A_3, A_7, was unbarred at the time we came to A_6 in our regular procedure—*this precisely because of our agreement to arrange the elements of G in non-ascending order of the periods.* But then A_6 should be barred, according to our rule of barring and leaving unbarred.

This contradiction establishes the independence of S, and so brings us to our goal of proving the theorem which is the subject of this note.

REPRESENTATIONS OF SEMISIMPLE LIE GROUPS. II

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Communicated by P. A. Smith, April 23, 1951

The object of this note is to announce some further results on representations of a connected semisimple Lie group on a Hilbert space. We shall omit all proofs and assume that the reader is familiar with the contents of an earlier note[1] (quoted henceforward as *RI*).

Let R and C denote the fields of real and complex numbers, respectively, and let G be a connected, simply connected, semisimple Lie group and $\mathfrak{g}_0$ its Lie algebra over R. Let $x \to Ad(x)(x \in G)$ denote the adjoint representation of G and let K be the complete inverse image in G of some maxi-

mal compact subgroup of $Ad(G)$. Then K is a closed connected subgroup of G. Let $\mathfrak{k}_0$ be the Lie algebra of K and let $X \to adX (X \in \mathfrak{g}_0)$ denote the adjoint representation of $\mathfrak{g}_0$. Put $B(X, Y) = sp(adXadY)(X, Y \in \mathfrak{g}_0)$. Let $\mathfrak{p}_0$ be the set of all elements $Y \in \mathfrak{g}_0$ such that $B(X, Y) = 0$ for all $X \in \mathfrak{k}_0$. Then $\mathfrak{g}_0 = \mathfrak{k}_0 + \mathfrak{p}_0$, $\mathfrak{k}_0 \cap \mathfrak{p}_0 = \{0\}$ and there exists an automorphism θ of $\mathfrak{g}_0$ over R such that $\theta(X + Y) = X - Y$ for $X \in \mathfrak{k}_0$, $Y \in \mathfrak{p}_0$. Let $\mathfrak{h}_{\mathfrak{p}_0}$ be a maximal abelian subspace of $\mathfrak{p}_0$. We extend $\mathfrak{h}_{\mathfrak{p}_0}$ to a maximal abelian subalgebra $\mathfrak{h}_0$ of $\mathfrak{g}_0$. Then $\mathfrak{h}_0 = \mathfrak{h}_{\mathfrak{k}_0} + \mathfrak{h}_{\mathfrak{p}_0}$ where $\mathfrak{h}_{\mathfrak{k}_0} = \mathfrak{h}_0 \cap \mathfrak{k}_0$. Let $\mathfrak{g}$ be the complexification of $\mathfrak{g}_0$ and let $\mathfrak{p}$, $\mathfrak{k}$, $\mathfrak{h}$, $\mathfrak{h}_{\mathfrak{p}}$, $\mathfrak{h}_{\mathfrak{k}}$ be the subspaces of $\mathfrak{g}$ spanned by $\mathfrak{p}_0$, $\mathfrak{k}_0$, $\mathfrak{h}_0$, $\mathfrak{h}_{\mathfrak{p}_0}$, $\mathfrak{h}_{\mathfrak{k}_0}$ respectively, over C. We extend the bilinear form B and the automorphism θ on $\mathfrak{g}$ by linearity over C. Choose bases $(H_1, \ldots, H_p)$ and $(H_{p+1}, \ldots, H_l)$, respectively, for $\mathfrak{h}_{\mathfrak{p}_0}$ and $\sqrt{-1}\,\mathfrak{h}_{\mathfrak{k}_0}$ over R. Let $\mathfrak{F}$ be the space of all linear functions on $\mathfrak{h}$. Given $\lambda \in \mathfrak{F}$ we can find a unique element $H_\lambda \in \mathfrak{h}$ such that $\lambda(H) = B(H, H_\lambda)$ for all $H \in \mathfrak{h}$. We shall say that λ is real if $H_\lambda \in \mathfrak{h}_{\mathfrak{p}_0} + \sqrt{-1}\,\mathfrak{h}_{\mathfrak{k}_0}$. Moreover if λ is real and $H_\lambda = \sum_{1 \leqslant i \leqslant l} c_i H_i (c_i \in R)$ we say that $\lambda > 0$ if $\lambda \neq 0$ and $c_j > 0$ where j is the least index $(1 \leqslant j \leqslant l)$ such that $c_j \neq 0$. We know that $\mathfrak{h}$ is a Cartan subalgebra of $\mathfrak{g}$ and $\theta\mathfrak{h} = \mathfrak{h}$. For every root α of $\mathfrak{g}$ with respect to $\mathfrak{h}$ let $X_\alpha \neq 0$ denote an element in $\mathfrak{g}$ such that $[H, X_\alpha] = \alpha(H)X_\alpha (H \in \mathfrak{h})$. Let P be the set of all roots $\alpha > 0$. For any $\lambda \in \mathfrak{F}$ let $\theta\lambda$ denote the linear function given by $\theta\lambda(H) = \lambda(\theta H)(H \in \mathfrak{h})$. Then if α is α root $\theta\alpha$ is also a root. Let P_+ be the set of all $\alpha \in P$ such that $\theta\alpha < 0$ and P the remaining set of positive roots. Iwasawa[2] has shown that $X_\alpha, X_{-\alpha} \in \mathfrak{k}$ for $\alpha \in P$. Put

$$\mathfrak{N} = \sum_{\alpha \in P_+} CX_\alpha, \quad \mathfrak{M} = \mathfrak{h}_{\mathfrak{k}} + \sum_{\alpha \in P} CX_\alpha + \sum_{\alpha \in P} CX_{-\alpha}.$$

Then $\mathfrak{N}$ is a nilpotent subalgebra of $\mathfrak{g}$ and $\mathfrak{M}$ is a subalgebra of $\mathfrak{k}$. Put $\mathfrak{N}_0 = \mathfrak{N} \cap \mathfrak{g}_0$, $\mathfrak{M}_0 = \mathfrak{M} \cap \mathfrak{k}_0$. Then $\mathfrak{g}_0 = \mathfrak{k}_0 + \mathfrak{h}_{\mathfrak{p}_0} + \mathfrak{N}_0$ where the sum is direct[2] and $[\mathfrak{M}, \mathfrak{N}] \subset \mathfrak{N}$, $[\mathfrak{M}, \mathfrak{h}_{\mathfrak{p}}] = \{0\}$. Moreover $\mathfrak{k}$ and $\mathfrak{M}$ are reductive algebras, i.e., they are the direct sums of their centers and their derived algebras which are semisimple. Let $\mathfrak{C}$ be the center of $\mathfrak{k}$. Put $\mathfrak{C}_0 = \mathfrak{C} \cap \mathfrak{g}_0$ and $\mathfrak{k}_0' = [\mathfrak{k}_0, \mathfrak{k}_0]$. Let D, K', A_+, M and N, respectively, be the analytic subgroups of G corresponding to $\mathfrak{C}_0$, $\mathfrak{k}_0'$, $\mathfrak{h}_{\mathfrak{p}_0}$, $\mathfrak{M}_0$ and $\mathfrak{N}_0$. Then M is closed and the mappings $(\gamma, u) \to \gamma u (\gamma \in D, u \in K')$ and $(v, h, n) \to vhn (v \in K, h \in A_+, n \in N)$ are analytic isomorphisms of DXK' with K and $K \times A_+ \times N$ with G, respectively.

Let Ω, Ω' and ω, respectively, be the set of all equivalence classes of finite-dimensional irreducible representations of K, K' and M. Then if $\mathfrak{D} \in \Omega$ and $\sigma \in \mathfrak{D}$ it is easily seen that $\sigma(K')$ is irreducible and $\sigma(M)$ is fully reducible. We denote by $\mathfrak{D}'$ the equivalence class of the irreducible representation of K' defined by σ. Also if $\delta \in \omega$ we say that $\delta < \mathfrak{D}$ if δ occurs in the reduction of $\sigma(M)$. Let $\alpha \in \delta \in \omega$. Then α defines α repre-

sentation β of $\mathfrak{M}_0$ and therefore of $\mathfrak{M}$. Let λ and μ be any two weights of β with respect to $\mathfrak{h}_2$. We extend λ and μ on $\mathfrak{h}$ by defining them to be zero on $\mathfrak{h}_3$. Then it is easily seen that $\lambda - \mu$ is a real linear function on $\mathfrak{h}$. Hence β has a weight λ such that $\lambda - \mu > 0$ for every other weight μ. Clearly λ depends on δ alone and we shall call it the highest weight of δ.

Let $\mathfrak{B}$ be the universal enveloping algebra of $\mathfrak{g}$ and let $\mathfrak{Z}$ be the center of $\mathfrak{B}$. Let $C[x]$ denote the ring of all commutative polynomials in l independent variables $x_1, \ldots, x_l$ with coefficients in C. We denote by β the isomorphic mapping of $C[x]$ into $\mathfrak{B}$ given by $\beta(x_1^{m_1}x_2^{m_2}\ldots x_l^{m_l}) = H_1^{m_1}\ldots H_l^{m_l}$. For any $z \in \mathfrak{Z}$ there exists a unique element[3] $\chi_x(z) \in C[x]$ such that $z - \beta(\chi_x(z)) \in \sum_{\alpha \in P} \mathfrak{B}X_\alpha$. Λ being any linear function on $\mathfrak{h}$ we denote by $\chi_\Lambda(z)$ the value of the polynomial $\chi_x(z)$ at $x_i = \Lambda(H_i) | \leq i \leq l$. Then the mapping $\chi_\Lambda : z \to \chi_\Lambda(z) (z \in \mathfrak{Z})$ is a homomorphism of $\mathfrak{Z}$ into C and conversely every homomorphism of $\mathfrak{Z}$ into C is of the form[3] χ_Λ for some $\Lambda \in \mathfrak{H}$. Let $\rho = {}^1/_2 \sum_{\alpha \in P} \alpha$ and let W be the Weyl group of $\mathfrak{g}$ with respect to $\mathfrak{h}$. Then if $\Lambda_1, \Lambda_2 \in \mathfrak{H}$, $\chi_{\Lambda_1} = \chi_{\Lambda_2}$ if and only if[3] $s(\Lambda_1 + \rho) = \Lambda_2 + \rho$ for some $s \in W$.

We denote by Ω_F the set of all $\mathfrak{D} \in \Omega$ for which there exists a finite-dimensional representation π of G such that $\mathfrak{D}'$ occurs in the reduction of $\pi(K')$.

THEOREM 1. *Let π be a quasisimple[1] representation of G on a Banach space with the infinitesimal character[4] χ. Suppose $\mathfrak{D}$ is an element in Ω_F such that $\mathfrak{D}$ occurs[5] in π. Then there exists a linear function Λ on $\mathfrak{h}$ and a $\delta \in \omega$ such that $\delta < \mathfrak{D}$ and $\chi = \chi_\Lambda$ and $\Lambda(H) = \lambda_\delta(H) (H \in \mathfrak{h}_2)$, where λ_δ is the highest weight of δ. Conversely suppose we are given a linear function Λ on $\mathfrak{h}$ and $\mathfrak{D} \in \Omega$ such that Λ coincides on $\mathfrak{h}_2$ with the highest weight λ_δ of some $\delta < \mathfrak{D}$ ($\delta \in \omega$). Then there exists a quasisimple irreducible representation π of G on a Hilbert space with the infinitesimal character χ_Λ such that $\mathfrak{D}$ occurs in π.*

Remarks.—It seems likely that the first part of the above theorem is actually true for all $\mathfrak{D} \in \Omega$ and not merely for $\mathfrak{D} \in \Omega_F$, but so far it has not been possible to prove this. Notice that if G is a complex semisimple group $\Omega = \Omega_F$ and so in this case the theorem holds without any restriction on $\mathfrak{D}$.

Let π_1 and π_2 be two quasisimple representations of G on the Hilbert spaces $\mathfrak{H}_1$ and $\mathfrak{H}_2$, respectively. For any $\mathfrak{D} \in \Omega$ let $\mathfrak{H}_{i,\mathfrak{D}}$ denote the set of all elements in $\mathfrak{H}_i$ which transform[1] under $\pi_i(K)$ according to $\mathfrak{D} (i = 1, 2)$. Put $\mathfrak{H}_i^0 = \sum_{\mathfrak{D} \in \Omega} \mathfrak{H}_{i,\mathfrak{D}}$. Then we get a representation π_i^0 of $\mathfrak{B}$ on $\mathfrak{H}_i^0$ such that

$$\pi_i^0(X)\psi = \lim_{t \to 0} \frac{1}{t} \{\pi_i(\exp tX)\psi - \psi\} \qquad (\psi \in \mathfrak{H}_i^0, X \in \mathfrak{g}_0, t \in R).$$

We say that π_1 and π_2 are infinitesimally equivalent if the representations π_1^0 and π_2^0 are algebraically equivalent, i.e., if there exists an isomorphism α of $\mathfrak{H}_1^0$ onto $\mathfrak{H}_2^0$ such that $\pi_2^0(b)\alpha\psi = \alpha\pi_1^0(b)\psi (b \in \mathfrak{B}, \psi \in \mathfrak{H}_1^0)$. Clearly if π_1 and π_2 are equivalent they are also infinitesimally equivalent. Conversely it can be shown that if π_1 and π_2 are both unitary, their infinitesimal equivalence implies their equivalence in the usual sense.

Let π be a quasisimple irreducible representation of G on a Hilbert space. We know (see Theorem 3 of *RI*) that dim $\mathfrak{H}_\mathfrak{D} < \infty$ for all $\mathfrak{D} \in \Omega$. Moreover, we may assume without loss of generality that the subspaces $\mathfrak{H}_\mathfrak{D}$ are all mutually orthogonal for distinct $\mathfrak{D}$. Let $E_\mathfrak{D}$ denote the orthogonal projection on $\mathfrak{H}$ on $\mathfrak{H}_\mathfrak{D}$. Put

$$\varphi_\mathfrak{D}^\pi(x) = sp(E_\mathfrak{D}\pi(x)E_\mathfrak{D}) \qquad (x \in G).$$

Then we can restate Theorem 7 of *RI* in a slightly improved form as follows.

THEOREM 2. *Let π_1, π_2 be irreducible quasisimple representations of G on two Hilbert spaces. Suppose that for some $\mathfrak{D} \in \Omega$ and $c \in C$, $\varphi_\mathfrak{D}^{\pi_1} = c\varphi_\mathfrak{D}^{\pi_2} \neq 0$. Then π_1 and π_2 are infinitesimally equivalent. Conversely if π_1 and π_2 are infinitesimally equivalent $\varphi_\mathfrak{D}^{\pi_1} = \varphi_\mathfrak{D}^{\pi_2}$ for all $\mathfrak{D} \in \Omega$.*

We have seen above that every element $x \in G$ can be written uniquely in the form $x = vhn (v \in K, h \in A_+, n \in N)$. For any $v \in K$ and $x \in G$ let v_x and $H(x, v)$ denote the unique elements in K and $\mathfrak{h}_{\mathfrak{k}_0}$, respectively, such that $xv = v_x(\exp H(x, v))n$ for some $n \in N$. Moreover let $\Gamma(v)$ denote the element in $\mathfrak{C}_0$ such that $v = (\exp \Gamma(v))u$ for some $u \in K'$. Put $\Gamma(x, v) = \Gamma(v_x) - \Gamma(v)$. Let Z be the center of G and let $z \to z^*$ denote the adjoint representation of G. Then $K \supset Z$ and K^* is compact. It is easily seen that $(va)_x = v_x a$, $H(x, va) = H(x, v)$, $\Gamma(x, va) = \Gamma(x, v) (a \in Z)$. Hence we may write $H(x, v) = H(x, v^*)$, $\Gamma(x, v) = \Gamma(x, v^*)$. Let dv^* denote the Haar measure on K^* such that $\int_{K^*} dv^* = 1$. For any $\mathfrak{D} \in \Omega$ let $\mu_\mathfrak{D}$ denote the linear function on $\mathfrak{C}$ such that

$$\sigma(\exp \Gamma) = e^{\mu_\mathfrak{D}(\Gamma)}\sigma(1) \qquad (\Gamma \in \mathfrak{C}_0)$$

for $\sigma \in \mathfrak{D}$. Also let $d(\mathfrak{D})$ denote the degree of σ.

THEOREM 3. *Let π be a quasisimple irreducible representation of G on a Hilbert space $\mathfrak{H}$ and let $\mathfrak{D}$ be an element in Ω such that $d(\mathfrak{D}) = 1$ and $\mathfrak{D}$ occurs[5] in π. Then dim $\mathfrak{H}_\mathfrak{D} = 1$ and there exists a linear function Λ on $\mathfrak{h}$ such that χ_Λ is the infinitesimal[4] character of π and*

$$\varphi_\mathfrak{D}^\pi(x) = \int_{K^*} e^{\mu_\mathfrak{D}(\Gamma(x, v^*))} e^{\Lambda(H(x, v^*))}\, dv^* \qquad (x \in G).$$

[1] Harish-Chandra, PROC. NATL. ACAD. SCI., **37**, 170–173 (1951).

[2] Iwasawa, K., *Ann. Math.*, **50**, 507–558 (1949).

[3] Harish-Chandra, *Trans. Am. Math. Soc.*, **70**, 28–96 (1951).

[4] The infinitesimal character of π was called simply the character of π in *RI*.

[5] This means that $\mathfrak{D}$ occurs in the reduction of $\pi(K)$.

REPRESENTATIONS OF SEMISIMPLE LIE GROUPS. III. CHARACTERS

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We shall adhere strictly to the notation of the preceding note.[1] Making use of an unpublished result of Chevalley one can prove the following theorem.

THEOREM 1. *Let π be a quasisimple irreducible representation of G on a Hilbert space $\mathfrak{H}$. Then there exists an integer N such that*

$$\dim \mathfrak{H}_{\mathfrak{D}} \leq N(d(\mathfrak{D}))^2$$

for any $\mathfrak{D} \in \Omega$.

Moreover if $\mathfrak{H}_{\mathfrak{D}} \neq \{0\}$ for some $\mathfrak{D} \in \Omega_r$ then it can be shown that we may take N equal to the order of the Weyl group W.

Let π be as above and let $C_c^\infty(G)$ denote the class of all complex-valued functions on G which are indefinitely differentiable everywhere and which vanish outside a compact set. Let A be a bounded operator on $\mathfrak{H}$. We say that A has a trace if for every complete orthonormal set[2] $(\psi_1, \psi_2, \ldots, \psi_n, \ldots)$ in $\mathfrak{H}$ the series $\sum_{i \geq 1} (\psi_i, A\psi_i)$ converges to a finite number independent of the choice of this orthonormal set. We denote this number by spA. Let A^* be the adjoint of A. We say that A is of the Hilbert-Schmidt class if AA^* has a trace.

THEOREM 2. *Let π be a quasisimple irreducible representation of G on a Hilbert space $\mathfrak{H}$. Then for any $f \in C_c^\infty(G)$ the operator*[3] $\int_G f(x)\pi(x)\,dx$ *has a trace. Put*

$$T_\pi(f) = sp\left(\int_G f(x)\pi(x)\,dx\right)$$

and for any $a \in G$ let ${}_af_a$ denote the function ${}_af_a(x) = f(a^{-1}xa)$ $(x \in G)$. Then T_π is a distribution in the sense of L. Schwartz[4] *and*

$$T_\pi({}_af_a) = T_\pi(f) \qquad (f \in C_c^\infty(G), a \in G).$$

We shall call the distribution T_π the character of the representation π.

THEOREM 3. *Let π_1 and π_2 be quasisimple irreducible representations of G on two Hilbert spaces. If $T_{\pi_1} = cT_{\pi_2}(c \in C)$ then π_1 and π_2 are infinitesimally equivalent. Conversely if π_1 and π_2 are infinitesimally equivalent $T_{\pi_1} = T_{\pi_2}$.*

Since for irreducible unitary representations infinitesimal equivalence is the same as ordinary equivalence, such a representation is completely determined within equivalence by its character.

THEOREM 4. *Let π be a quasisimple irreducible representation of G and let f be any measurable function on G such that f vanishes outside a compact set and $\int_G |f(x)|^2\,dx < \infty$. Then the operator $\int_G f(x)\pi(x)\,dx$ is of the Hilbert-Schmidt class.*

For any $X \in \mathfrak{g}_0$ and $f \in C_c^\infty(G)$ put

$$(^*Xf)(x) = \left\{\frac{d}{dt} f(\exp(-tX)x)\right\}_{t=0}.$$

Then the mapping $X \to {}^*X$ is a representation of $\mathfrak{g}_0$ on $C_c^\infty(G)$ which can be extended to a representation $b \to {}^*b$ ($b \in \mathfrak{B}$) of $\mathfrak{B}$. Let φ be the anti-automorphism of $\mathfrak{B}$ such that $\varphi(X) = -X$ ($X \in \mathfrak{g}$). If T is any distribution on G we define bT ($b \in \mathfrak{B}$) as follows:

$$bT(f) = T(^*(\varphi(b))f) \qquad (f \in C_c^\infty(G)).$$

Moreover for any function f on G we denote by ${}_yf$, f_z and ${}_yf_z$ ($y, z, \in G$) the functions

$${}_yf(z) = f(y^{-1}x),\ f_z(x) = f(xz),\ {}_yf_z(x) = f(y^{-1}xz)\ (z \in G).$$

Let Z denote the center of G and let π be a quasisimple irreducible representation of G on a Hilbert space. Let χ be the infinitesimal character of π and let η be the homomorphism of Z into C such that $\pi(a) = \eta(a)\pi(1)$ ($a \in Z$). Then if T_π is the character of π it is easily seen that

$$zT_\pi = \chi(z)T_\pi \qquad (z \in \mathfrak{Z})$$

$$T_\pi({}_yf_y) = T_\pi(f),\ T_\pi(f_a) = \eta(a)T_\pi(f)\ (f \in C_c^\infty(G),\ y \in G,\ a \in Z).$$

Now put[5] $M_1 = MZ$ and let $x \to x^*$ denote the adjoint representation of G. Put $(x)^{y^*} = yxy^{-1}$ ($x, y \in G$) and let $C_c(G)$ denote the class of all continuous functions on G which vanish outside a compact set. Let α and g be continuous functions[5] on A_+ and M_1, respectively. Put[6]

$$T(f) = \int f((n^{-1}h^{-1}m^{-1})^{u^*})\alpha(h)g(m)\,dm\,dh\,dn\,du^* \qquad (f \in C_c(G))$$

Here dm, dh, dn, du^* are the left invariant Haar measures on M_1, A_+, N, K^*, respectively, and the integral extends over $K^* \times M_1 \times A_+ \times N$. It can be shown that $T(f) = T({}_yf_y)$ ($y \in G$). Let σ be an irreducible representation of M_1 on a finite-dimensional Hilbert space U. Let δ be the equivalence class of the representation σ_0 of M defined by σ. Let $\psi_0 \neq 0$ i e an element in U belonging to the highest weight λ_δ of σ_0 and let η be the homomorphism of Z into C such that $\sigma(a) = \eta(a)\sigma(1)$ ($a \in Z$). Put $g(m) = (\psi_0, \sigma(m^{-1})\psi_0)$ ($m \in M_1$) and $\alpha(h) = e^{-(\nu+2\rho)(\log h)}$ where ν is a linear function on $\mathfrak{h}_\mathfrak{B}$ and $\log h$ is the unique element in $\mathfrak{h}_{\mathfrak{p}_0}$ such that $\exp(\log h) = h$. If we regard T as a distribution it is easily seen that

$$zT = \chi_\Lambda(z)T \qquad (z \in \mathfrak{Z})$$

where $\Lambda(H_1 + H_2) = \nu(H_1) + \lambda_\beta(H_2)$ $(H_1 \in \mathfrak{h}_\nu, H_2 \in \mathfrak{h}_\beta)$. Moreover

$$T(f_a) = \eta(a)T(f) \qquad (a \in Z, f \in C_c(G))$$

and it is easy to check that

$T(f) = \int f((n^{-1}h^{-1}m^{-1})^{u^*})e^{-(\nu+2\rho)(\log h)}\,\xi(m^{-1})\,dm\,dh\,dn\,du^*$ $(f \in C_c(G))$ where

$$\xi(m) = \frac{1}{d(\delta)}|\psi_0|^2 \; sp\sigma(m)$$

and $d(\delta)$ is the degree of σ. Let A_- be the analytic subgroup of M corresponding to $\mathfrak{h}_{\nu_0}$. Put $A = A_+A_-$ and $A_1 = AZ$. Let V be the set of all elements in G which can be written in the form xyx^{-1} with $x \in G$ and $y \in A_1N$. We shall say that an element $y \in V$ is regular if $y = xhx^{-1}$ for some $x \in G$ and $h \in A_1$ and y^* has exactly[5] l eigen-values equal to 1. Let V_0 be the set of all elements in V which are regular. Then V_0 is open in G and V is the closure of V_0. Let W_0 be the subgroup of the Weyl group W consisting of those elements $s \in W$ for which there exists an $x \in G$ such that $sH = x^*H$ for all $H \in \mathfrak{h}$. It is easily seen that every $s \in W_0$ leaves both $\mathfrak{h}_\nu$ and $\mathfrak{h}_\beta$ invariant. Put

$$\Delta^-(H) = \prod_{\alpha \in P_-}(e^{\frac{1}{2}\alpha(H)} - e^{-\frac{1}{2}\alpha(H)}),\ \Delta^+(H) = \prod_{\alpha \in P_+}(e^{\frac{1}{2}\alpha(H)} - e^{-\frac{1}{2}\alpha(H)}) \qquad (H \in \mathfrak{h})$$

and define $\epsilon(s) = \pm 1$ $(s \in W_0)$ in such a way that

$$\Delta^-(sH) = \epsilon(s)\Delta^-(H)$$

for all $H \in \mathfrak{h}_\nu$. In particular if P_- is empty $\epsilon(s) = 1$ for all $s \in W_0$. Consider the function $\Theta_{\Lambda,\eta}$ on V_0 defined as follows:

$$\Theta_{\Lambda,\eta}(y) = \eta(\gamma)\frac{\sum_{s \in W_0}\epsilon(s)e^{s(\Lambda+\rho)(H_1+H_2)}}{\Delta^-(H_1)\Delta^+(H_1+H_2)} \qquad (y \in V_0)$$

where $y = x(\gamma \exp(H_1 + H_2))x^{-1}$ for some $x \in G$, $\gamma \in Z$, $H_1 \in \mathfrak{h}_{\nu_0}$ and $H_2 \in \mathfrak{h}_{\beta_0}$. It can be shown that in spite of the ambiguity in the choice of γ, H_1 and H_2, $\Theta_{\Lambda,\eta}$ is well defined on V_0. We extend $\Theta_{\Lambda,\eta}$ on G by defining it to be zero outside V_0. Then it can be proved that[6]

$$T(f) = \int_G f(x)\,\Theta_{\Lambda,\eta}(x)\,dx \qquad (f \in C_c(G))$$

provided the Haar measure dx on G is suitably normalized. Let $s_1 = 1$ $s_2, \ldots, s_r$ be a maximal set of distinct elements in the Weyl group W with the following properties:

(I) Let $\Lambda_i = s_i(\Lambda + \rho) - \rho$ $1 \leq i \leq r$. Then $\Lambda_i + \rho \neq s(\Lambda_j + \rho)$ if $i \neq j$ $(1 \leq i, j \leq r)$ and $s \in W_0$.

(II) For each i $(1 \leq i \leq r)$ there exists a $\delta_i \in \omega$ such that Λ_i coincides on

$\mathfrak{h}_\nu$ with the highest weight[5] λ_{δ_i} of δ_i. Moreover if $\sigma \in \delta_i$ and $\gamma \in M \cap Z$

$$\sigma(\gamma) = \eta(\gamma)\sigma(1).$$

Put $\Theta_i = \Theta_{\Lambda_i, \eta}$ $1 \leq i \leq r$ and $T_i(f) = \int_G f(x)\Theta_i(x)\,dx \qquad (f \in C_c(G))$. Then we see that the distributions T_i $1 \leq i \leq r$ are solutions of the equations,

$$zT = \chi_\Lambda(z)T \qquad (z \in \mathfrak{Z})$$

$$T(_y f_y) = T(f),\ T(f_a) = \eta(a)T(f)\ (f \in C_c^\infty(G),\ y \in G,\ a \in Z).$$

We have seen above that if π is any quasisimple irreducible representation of G such that χ_Λ is the infinitesimal character of π and $\pi(a) = \eta(a)\pi(1)$ $(a \in Z)$, then its character T_π is also a solution of the above equations. Hence one might hope that in most cases $T\pi$ would be a linear combination of T_i $1 \leq i \leq r$.

In conclusion I should like to thank Professor C. Chevalley for his help and advice on several questions connected with the results of this note.

[1] "Representations of semisimple Lie groups. II," PROC. NATL. ACAD. SCI., **37**, 362 (1951), quoted hereafter as *RII*.

[2] Since π is irreducible it follows easily that $\mathfrak{H}$ is separable.

[3] Here dx denotes the left invariant Haar measure on G.

[4] Schwartz, L., *Theorie des distributions*, Hermann, Paris, 1950.

[5] See *RII* for the meaning of the various symbols.

[6] Compare with Gelfand I. M., and Naimark M. A., *Izvestiya Akad. Nauk SSR*, Ser. Mat., 1947, vol. 11, pp. 411–504; *Mat. Sbornik N. S.*, 1947, vol. 21 (63), pp. 405–434; *Doklady Akad. Nauk SSSR* (*N. S.*), 1948, vol. 61, pp. 9–11.

ON THE NUMERICAL COMPUTATION OF MAPPING FUNCTIONS BY ORTHOGONALIZATION

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By means of a technique introduced by Bergman (reference 1, and literature quoted there), it is possible to reduce the computation of a large number of conformal mapping functions and harmonic domain functions to the orthonormalization of given complete sets of functions with respect to suitably defined inner products.[1-3] While the convergence of the expansions obtained in this way is assured, the general method gives no indication as to the quality of the convergence. No estimate for the error committed in replacing the infinite expansions by finite partial sums is given and this, of course, represents a serious drawback as far as the practical application of the Bergman method is concerned.

It is the purpose of this note to show how in the case of one particular domain function this gap may be filled in. The function in question is the Szegö kernel $K(z, \zeta)$[2, 5] of an analytically bounded plane domain D. $K(z, \zeta)$ is defined by the expansion

$$K(z, \zeta) = \sum_{\nu=1}^{\infty} u_\nu(z)\overline{u_\nu(\zeta)}, \quad z \in D, \zeta \in D, \tag{1}$$

where the analytic functions $u_\nu(z)$ are regular and single-valued in D and are orthonormalized by the conditions

$$\int_C u_\nu(z)\overline{u_\mu(z)}\, ds = \delta_{\nu\mu}, \; ds = |dz|, \tag{2}$$

where C is the boundary of D. Furthermore, the set $\{u_\nu(z)\}$ has to be complete in the sense that any function $f(z)$ which is regular and single-valued in D and possesses a finite integral $\int_C |f(z)|^2\, ds$ may be approximated by linear combinations of the $u_\nu(z)$ in such a way as to make the integral

$$\int_C \Big| f(z) - \sum_{\nu=1}^{n} a_\nu u_\nu(z) \Big|^2 ds$$

arbitrarily small. Under fairly general conditions, the set $\{u_\nu(z)\}$ may be obtained by orthonormalizing a certain simple set of rational functions.[1] In particular, if D is simply connected and finite and, moreover, is such that its complement is a closed domain, then a suitable set $\{u_\nu(z)\}$ may be constructed by orthonormalizing the powers $1, z, z^2, \ldots$ by means of the conditions (2)[6].

The function $K(z, \zeta)$ is intimately related to the solution of the problem $|f'(\zeta)| = \max.$, where $f(z)$ is regular and single-valued and $|f(z)| < 1$ in D and $\zeta \in D$.[2, 4] As shown in reference 2, the maximum in question is $2\pi K(\zeta, \zeta)$. If D is simply connected, there exists a simple relation between $K(z, \zeta)$ and the function $w = f(z) = f(z, \zeta)$ which maps D conformally onto the unit circle $|w| < 1$ in such a way that $f(\zeta) = 0, f'(\zeta) > 0$. We have[5]

$$f'(z, \zeta) = \frac{2\pi}{K(\zeta, \zeta)} K^2(z, \zeta),$$

which shows that the conformal mapping of simply connected domains may be reduced to the computation of the function $K(z, \zeta)$.

In order to estimate the error committed in replacing the expansion (1) by its first n terms, it is sufficient to do so for $z = \zeta$. Indeed, we have

$$K(z, \zeta) = K_n(z, \zeta) + R_n(z, \zeta), \tag{3}$$

where

$$K_n(z, \zeta) = \sum_{\nu=1}^{n} u_\nu(z)\overline{u_\nu(\zeta)} \tag{4}$$

and

$$R_n(z, \zeta) = \sum_{\nu=n+1}^{\infty} u_\nu(z)\overline{u_\nu(\zeta)}.$$

Hence, by the Schwarz inequality,

$$|R_n(z, \zeta)|^2 \leq R_n(z, z)R_n(\zeta, \zeta),$$

and therefore

$$|K(z, \zeta) - K_n(z, \zeta)|^2 \leq R_n(z, z)R_n(\zeta, \zeta). \tag{5}$$

It was shown in reference 2 that there exists a function $L(z, \zeta)$ which is single-valued and, except for a pole with the meromorphic part $[2\pi(z - \zeta)]^{-1}$, regular in D, and which is connected with $K(z, \zeta)$ by the boundary relation

$$\overline{L(z, \zeta)}\, ds = -iK(z, \zeta)\, dz, \quad z \in C; \tag{6}$$

moreover, both $K(z, \zeta)$ and $L(z, \zeta)$ are regular on C. If a_ν denotes the νth Fourier coefficient of $L(z, \zeta) - [2\pi(z - \zeta)]^{-1}$ with respect to the orthonormal set $\{u_\nu(z)\}$, that is,

$$a_\nu = \int_C \left[L(z, \zeta) - \frac{1}{2\pi(z - \zeta)}\right] \overline{u_\nu(z)}\, ds, \tag{7}$$

it follows from the completeness of the set that

$$\sum_{\nu=1}^{\infty} |a_\nu|^2 = \int_C \left|L(z, \zeta) - \frac{1}{2\pi(z - \zeta)}\right|^2 ds. \tag{8}$$

Now we have, in view of (7) and (6),

$$a_\nu = -i \int_C K(z, \zeta)u_\nu(z)\, dz - \frac{1}{2\pi} \int_C \frac{\overline{u_\nu(z)}}{z - \zeta}\, ds.$$

By Cauchy's theorem, the first integral vanishes, whence

$$a_\nu = -\frac{1}{2\pi} \int_C \frac{\overline{u_\nu(z)}}{z - \zeta}\, ds \tag{9}$$

From (8), we obtain

$$\sum_{\nu=1}^{\infty} |a_\nu|^2 = \int_C |L(z, \zeta)|^2\, ds - 2Re\left\{\frac{1}{2\pi} \int_C \frac{\overline{L(z, \zeta)}}{z - \zeta}\, ds\right\} + \frac{1}{4\pi^2} \int_C \frac{ds}{|z - \zeta|^2}.$$

Hence, by (6) and the residue theorem,

$$\sum_{\nu=1}^{\infty} |a_\nu|^2 = -i \int_C L(z,\zeta)K(z,\zeta)\,dz - 2Re\left\{\frac{1}{2\pi i}\int_C \frac{K(z,\zeta)}{z-\zeta}\,dz\right\} + \frac{1}{4\pi^2}\int_C \frac{ds}{|z-\zeta|^2} = K(\zeta,\zeta) - 2Re\{K(\zeta,\zeta)\} + \frac{1}{4\pi^2}\int_C \frac{ds}{|z-\zeta|^2}.$$

Since $K(\zeta, \zeta)$ is positive, it thus follows that

$$K(\zeta,\zeta) = \frac{1}{4\pi^2}\int_C \frac{ds}{|z-\zeta|^2} - \sum_{\nu=1}^{\infty} |a_\nu|^2 \leq \frac{1}{4\pi^2}\int_C \frac{ds}{|z-\zeta|^2} - \sum_{\nu=1}^{n} |a_\nu|^2, \tag{10}$$

where a_ν is defined by (9). On the other hand, (1) yields

$$K(\zeta,\zeta) = \sum_{\nu=1}^{\infty} |u_\nu(\zeta)|^2 \geq \sum_{\nu=1}^{n} |u_\nu(\zeta)|^2 = K_n(\zeta,\zeta). \tag{11}$$

Combining (10) and (11) and observing (5), we thus arrive at the following result.

If $K(z, \zeta)$ is the Szegö kernel and $K_n(z, \zeta)$ is the partial sum (4), *then*

$$|K(z,\zeta) - K_n(z,\zeta)|^2 \leq S_n(z)S_n(\zeta),$$

where

$$S_n(t) = \frac{1}{4\pi^2}\int_C \frac{ds}{|z-t|^2} - \sum_{\nu=1}^{n} [|a_\nu(t)|^2 + |u_\nu(t)|^2],$$

and $a_\nu(t)$ is defined by

$$a_\nu(t) = \frac{1}{2\pi}\int_C \frac{\overline{u_\nu(z)}}{z-t}\,ds.$$

If the set $\{u_\nu(z)\}$ is complete, then $S_n(t) \to 0$ for $n \to \infty$.

[1] Bergman, S., *The Kernel Function and Conformal Mapping*, New York, 1950.

[2] Garabedian, P., "Schwarz Lemma and the Szegö Kernel Function," *Trans. Am. Math. Soc.*, **67**, 1–35 (1949).

[3] Nehari, Z., "The Kernel Function and Canonical Conformal Maps," *Duke Math. J.*, **16**, 165–178 (1949).

[4] Nehari, Z., "On Bounded Analytic Functions," *Proc. Am. Math. Soc.*, **1**, 268–275 (1950).

[5] Szegö, G., "Ueber orthogonale Polynome, die zu einer gegebenen Kurve der komplexen Ebene gehoeren," *Math. Zeits.*, **9**, 218–270 (1921).

[6] In the case in which D is simply connected and $K(z, \zeta)$ is approximated by polynomials, an estimate of the order of magnitude of the remainder can be obtained by means of the method indicated by J. L. Walsh in *Interpolation and Approximation*, New York, 1935, § 6.9.

FINITE FOURIER SERIES AND CYCLOTOMY

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The method of finite Fourier series is a powerful tool for attacking many problems in the theory of numbers. These series are intimately related to certain types of exponential and trigonometric sums. They have been employed for number theoretic purposes by G. Eisenstein[1] and H. A. Rademacher.[2]

In order to state the basic formulas involved in the method we use the following notation. Let μ be a non-negative integer, m a positive integer and $\alpha = e^{2\pi i/m}$ an mth root of unity. An arbitrary function $f(\alpha^\mu)$ is periodic in μ with respect to the modulus m. It may therefore be expanded into a finite Fourier series of the form

$$f(\alpha^\mu) = \sum_{j=0}^{m-1} g(j)\alpha^{\mu j} \quad (\mu = 0, 1, \ldots, m-1). \tag{1}$$

Indeed the system of linear equations (1) in the unknowns $g(j)$ has a non-vanishing (Vandermonde) determinant and has therefore a unique solution. The orthogonality relation

$$\sum_{j=0}^{m-1} \alpha^{aj}\alpha^{-bj} = \begin{cases} m & (a \equiv b \pmod m), \\ 0 & (a \not\equiv b \pmod m), \end{cases}$$

enables us to determine the finite Fourier coefficients $g(k)$ explicitly by means of the formula

$$g(k) = \frac{1}{m}\sum_{\mu=0}^{m-1} f(\alpha^\mu)\alpha^{-\mu k} \qquad (k = 0, 1, \ldots, m-1). \tag{2}$$

The finite Parseval relation is given by

$$\sum_{j=0}^{m-1} g(j)\bar{g}(j+k) = \frac{1}{m}\sum_{\mu=0}^{m-1} |f(\alpha^\mu)|^2\alpha^{\mu k} \qquad (k = 0, 1, \ldots, m-1), \tag{3}$$

where $\bar{g}(j)$ denotes the complex conjugate of $g(j)$.

To illustrate the method we now mention an application which does not seem to have been noted previously. For an integer a not divisible by an odd prime p let $S_p(a)$ denote the Kloosterman sum

$$S_p(a) = \sum_{h=1}^{p-1} e^{2\pi i(ah + \bar{h})/p}$$

where $\bar{h}$ denotes any solution of the congruence $h\bar{h} \equiv 1 \pmod p$. Expand-

ing the product $S_p(a)S_p(b)$ into a finite Fourier series and making use of (2) we may deduce the relation

$$S_p(a)S_p(b) = \sum_{h=1}^{p-1} e^{2\pi i((a+b)h+\bar{h})/p} S_p(abh^2), \tag{4}$$

provided $a \not\equiv b \pmod{p}$. If $a \equiv b \pmod{p}$ the right member of the last equation should be augmented by p. Formula (4) is due to Davenport.[3] His proof is along completely different lines.

In the present paper we employ multiple finite Fourier series to obtain generalizations of formulas (1), (2) and (3). We then apply our results to a number of problems in the theory of cyclotomy. Our methods are extensions of methods used by Vandiver[4] in a series of papers. Detailed proofs will be published elsewhere.

For $i = 1, 2, \ldots, s$ let μ_i be a non-negative integer, m_i a positive integer and $\alpha_i = e^{2\pi i/m_i}$ an m_ith root of unity. Consider a function $f(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})$ defined by means of the sum

$$f(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) = \sum_{j_1, j_2, \ldots, j_s} g(j_1, j_2, \ldots, j_s) \prod_{i=1}^{s} \alpha_i^{\mu_i j_i},$$

where the j_i range independently over $0, 1, \ldots, m_i - 1$. The value of the multiple finite Fourier coefficient $g(k_1, k_2, \ldots, k_s)$ is given by the relation

$$g(k_1, k_2, \ldots, k_s) \prod_{i=1}^{s} m_i = \sum_{\mu_1, \mu_2, \ldots, \mu_s} f(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) \prod_{i=1}^{s} \alpha_i^{-\mu_i k_i},$$

where the μ_i range independently over $0, 1, \ldots, m_i - 1$. The finite Parseval relation is

$$\prod_{i=1}^{s} m_i \sum_{j_1, j_2, \ldots, j_s} g(j_1, \ldots, j_s)\bar{g}(j_1 + k_1, \ldots, j_s + k_s) =$$

$$\sum_{\mu_1, \mu_2, \ldots, \mu_s} \left|f(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})\right|^2 \prod_{i=1}^{s} \alpha_i^{\mu_i k_i}. \tag{5}$$

Let p be an odd prime and $F(p^n)$ or K the finite field of order p^n. Let g be a generator of the cyclic group formed by the non-zero elements of K under multiplication. For a non-zero element a of K let ind a be defined by means of the equation $g^{\text{ind } a} = a$. In the theory of cyclotomy the generalized Jacobi sum $\psi(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})$ of Vandiver[5] plays a fundamental role. This sum is defined for $s > 1$ by

$$\psi(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) = \sum_{a_1, a_2, \ldots, a_{s-1}} \alpha_s^{\mu_s \text{ ind } A} \prod_{i=1}^{s-1} \alpha_i^{\mu_i \text{ ind } a_i} \tag{6}$$

and for $s = 1$ by $\psi(\alpha^{\mu}) = 1$. In this definition the integers m_i are restricted to be divisors of $p^n - 1$ so that we may write $p^n - 1 = m_i m_i'$, $i = 1, 2, \ldots, s$. The elements a_i range independently over each element

of K, and the element A is defined by means of the equation $A = 1 - a_1 - a_2 - \ldots - a_{s-1}$. The convention is also made that $\alpha_i^{\mu_i \text{ ind } (0)} = 0$.

Closely related to the generalized Jacobi sum is the generalized Lagrange resolvent[6] defined by

$$\tau(\alpha^\mu) = \sum_{a \in K} \alpha^{\mu \text{ ind } a} \zeta^{\text{tr}(a)}, \tag{7}$$

where $\zeta = e^{2\pi i/p}$ and $\text{tr}(a)$ is the trace of a in $F(p^n)$ with respect to $F(p)$. From the definition of $\text{tr}(a)$ it follows at once that $\text{tr}(a) + \text{tr}(b) = \text{tr}(a + b)$. The following relation is also known[6]

$$\sum_{a \in K} \zeta^{\text{tr}(ab)} = \begin{cases} p^n & (b = 0), \\ 0 & (b \neq 0), \end{cases} \tag{8}$$

where b is an element of K. The function $\tau(\alpha^\mu)$ has been the starting point of a number of deep investigations in finite field theory.[7] Its most important property[6] is given by

$$\tau(\alpha^\mu)\tau(\alpha^{-\mu}) = \alpha^{\mu \text{ ind } (-1)} p^n, \tag{9}$$

provided $\alpha^\mu \neq 1$. This function may also be employed to study Eisenstein sums.[8] We give here an application of this type. A prime p of the form $8n + 3$ is also a prime in the algebraic field of Gauss numbers. The field $F(p^2)$ may be represented by means of the residue classes with respect to p of the ring of algebraic integers in the Gauss field. Let $\alpha = e^{2\pi i/8}$ be an eighth root of unity. Then the Eisenstein sum

$$\theta(\alpha) = \sum_{v=0}^{p-1} \alpha^{\text{ind } (1 + iv)}$$

has the property

$$\theta(\alpha)\theta(\alpha^{-1}) = p.$$

This result is due to Eisenstein.[8] It may be derived from (7), (8) and (9) by separating the Gauss integers $u + iv$ into classes according to the quadratic character of u with respect to p.

We now return to the ψ-function defined in (6). For an integer r, $0 \leqq r \leqq s$, we define the function $\psi_r(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})$ as a ψ-number for which $\mu_i \not\equiv 0 \pmod{m_i}$ for r values of i and $\mu_i \equiv 0 \pmod{m_i}$ for the remaining $s - r$ values of i. From the results of Faircloth and Vandiver[6] on the ψ-function the following formulas follow immediately:

$$|\psi_r|^2 = p^{n(r-2)} \qquad (2 \leqq r \leqq s, \ \prod_{i=1}^{s} \alpha_i^{\mu_i} = 1), \tag{10a}$$

$$|\psi_r|^2 = p^{n(r-1)} \qquad (1 \leqq r \leqq s, \ \prod_{i=1}^{s} \alpha_i^{\mu_i} \neq 1). \tag{10b}$$

Note that r cannot equal 1 under the condition imposed in (10a). The case $r = 0$ is not covered in formulas (10a) and (10b). In this case, however, we may use (8) to establish directly that

$$\psi_0 = \psi(1, 1, \ldots, 1) = \frac{1}{p^n}\left((p^n - 1)^s + (-1)^{s+1}\right). \qquad (11)$$

The ψ-function has the alternative representation[5]

$$\psi(\alpha_1^{\mu_1}, \ldots, \alpha_1^{\mu_1}) = \epsilon(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) \sum_{j_1, j_2, \ldots, j_s} (j_1, j_2, \ldots, j_s) \prod_{i=1}^{s} \alpha_i^{\mu_i j_i}, \quad (12)$$

where

$$\epsilon = \epsilon(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) = \alpha_1^{\mu_1 \operatorname{ind}(-1)} \ldots \alpha_s^{\mu_s \operatorname{ind}(-1)}.$$

The symbol $(j_1, j_2, \ldots, j_s)$ represents the number of solutions in $\gamma_1, \gamma_2, \ldots, \gamma_s$ of the equation

$$1 + g^{j_1 + m_1\gamma_1} + g^{j_2 + m_2\gamma_2} + \ldots + g^{j_s + m_s\gamma_s} = 0,$$

for a fixed set of numbers $j_1, j_2, \ldots, j_s$ with the j_t in the range $0, 1, \ldots, m_t - 1$ and the γ_t in the range $0, 1, \ldots, m_t' - 1$. Since $g^{m_i m_i'/2} = g^{(p^n - 1)/2} = -1$ in K, we see that $\alpha_i^{\mu_i \operatorname{ind}(-1)} = \alpha_i^{\mu_i m_i m_i'/2} = \pm 1$, the sign depending on the value of m_i. Hence $\epsilon = \pm 1$. We put

$$\psi_r(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s}) = \epsilon(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})\Psi_r(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})$$

and note that Ψ_r has with ψ_r the properties (10a) and (10b). The sum in the right member of (12) may be regarded as a multiple finite Fourier series expansion of $\Psi(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})$. The Parseval relation (5) yields

$$\prod_{i=1}^{s} m_i \sum_{j_1, j_2 \ldots, j_s} (j_1, \ldots, j_s)(j_1 + k_1, \ldots, j_s + k_s) = \sum_{\mu_1, \mu_2, \ldots, \mu_s} \left|\Psi(\alpha_1^{\mu_1}, \ldots, \alpha_s^{\mu_s})\right|^2 \prod_{i=1}^{s} \alpha_i^{-\mu_i k_i}. \quad (13)$$

We denote by $S(k_1, k_2, \ldots, k_s)$ the left member of (13). In order to evaluate the right member of (13) we find it convenient to introduce certain auxiliary sums. Let

$$E = E_r = E(k_{i_1}, k_{i_2}, \ldots, k_{i_r}) \qquad (0 \leqq r \leqq s)$$

denote one of the 2^s sets of k's (including the vacuous set) which may be selected from a set of s non-negative integers $k_1, k_2, \ldots, k_s$. We first introduce the function $\delta(\alpha_i^{k_i})$ defined by

$$\delta(\alpha_i^{k_i}) = \sum_{\mu_i = 1}^{m_i - 1} \alpha_i^{-\mu_i k_i} = \begin{cases} m_i - 1 & (k_i \equiv 0 \pmod{m_i}), \\ -1 & (k_i \not\equiv 0 \pmod{m_i}). \end{cases} \quad (14)$$

In terms of the δ-function we define for $1 \leqq r \leqq s$ the function

$$\lambda_r(E_s) = \lambda_r(\alpha_1^{k_1}, \ldots, \alpha_s^{k_s}) = \sum_{E_r} \delta(\alpha_{t_1}^{k_{t_1}}) \ldots \delta(\alpha_{t_r}^{k_{t_r}}), \qquad (15)$$

where the sum extends over the $\binom{s}{r}$ sets E_r. In other words, λ_r is the rth elementary symmetric function of the numbers $\delta(\alpha_1^{k_1}), \ldots, \delta(\alpha_s^{k_s})$. In particular, $\lambda_1(E_s) = \delta(\alpha_1^{k_1}) + \ldots + \delta(\alpha_s^{k_s})$ and $\lambda_s(E_s) = \delta(\alpha_1^{k_1}) \ldots \delta(\alpha_s^{k_s})$.

We next introduce the function

$$\rho(E_s) = \rho(\alpha_1^{k_1}, \ldots, \alpha_s^{k_s}) = \sum \alpha_1^{-\mu_1 k_1} \ldots \alpha_s^{-\mu_s k_s}, \qquad (16)$$

where the sum extends over all the sets $\mu_1, \mu_2, \ldots, \mu_s$ for which $\prod_{i=1}^{s} \alpha_i^{\mu_i} = 1$ and $1 \leqq \mu_i \leqq m_i - 1$, $i = 1, 2, \ldots, s$. Finally we define for $1 \leqq r \leqq s$ the function

$$\phi_r(E_s) = \phi_r(\alpha_1^{k_1}, \ldots, \alpha_s^{k_s}) = \sum_{E_r} \rho(E_r). \qquad (17)$$

Note in particular that each term of $\phi_1(E_s) = \rho(\alpha_1^{k_1}) + \ldots + \rho(\alpha_s^{k_s})$ is a vacuous sum.

Using (10a), (10b) and (11) together with the notation in (15) and (17) we may transform (13) into

$$S(k_1, k_2, \ldots, k_s) = \frac{1}{p^{2n}} ((p^n - 1)^s + (-1)^{s+1})^2 + \sum_{r=1}^{s} p^{n(r-1)} \lambda_r(E_s) - \sum_{r=2}^{s} (p^{n(r-1)} - p^{n(r-2)}) \phi_r(E_s). \qquad (18)$$

To evaluate the ρ-function defined in (16) we first show that it may be written in the form

$$\rho(E_s) = \frac{1}{m} \sum_{h=0}^{m-1} \delta(\alpha_1^{h-k_1}) \ldots \delta(\alpha_s^{h-k_s}), \qquad (19)$$

where m is the least common multiple of the numbers $m_1, m_2, \ldots, m_s$. The solution is then obtained in terms of a function $d(E_r)$ defined in the following manner. Let $d_{ij} = (k_i, k_j)$ denote the greatest common divisor of k_i and k_j; let $m(E_r) = [m_{t_1}, \ldots, m_{t_r}]$ denote the least common multiple of a set of r m's. For the vacuous set E_0 put $d(E_0) = 1$. For the set $E(k_i)$ consisting of a single member put $dE(k_i) = 1$. For $2 \leqq r \leqq s$ put $d(E_r) = m_{t_1}, m_{t_2} \ldots m_{t_r}/m(E_r)$, provided that $k_i - k_j \equiv 0 \pmod{d_{ij}}$ for every pair k_i, k_j belonging to E_r. Otherwise put $d(E_r) = 0$. Then we have the identity

$$\rho(E_s) = \sum_{r=0}^{s} \sum_{E_r} (-1)^{s-r} d(E_r). \tag{20}$$

To derive (20) from (19) we employ a combinatorial argument in conjunction with the Chinese remainder theorem. Note that $\rho(E_s) = 0$ when the numbers $m_1, m_2, \ldots, m_s$ are prime each to each.

Formulas (14), (15), (17), (18) and (20) provide a solution of the problem of evaluating $S(k_1, k_2, \ldots, k_s)$. The case $s = 2$ yields immediately results due to Vandiver.[4] Among the numerous special cases for arbitrary s the following formulas are representative. If $m_1, m_2, \ldots, m_s$ are prime each to each, then

$$S(0, 0, \ldots, 0) = \frac{1}{p^{2n}}((p^n - 1)^s + (-1)^{s+1})^2 + \frac{1}{p^n}(-1 + \prod_{i=1}^{s}(1 + (m_i - 1)p^n)).$$

If $m_1, m_2, \ldots, m_s$ are prime each to each and $k_i \not\equiv 0 \pmod{m_i}$, $i = 1, 2, \ldots, s$, then

$$S(k_1, k_2, \ldots, k_s) = \frac{1}{p^{2n}}((p^n - 1)^s + (-1)^{s+1})^2 + \frac{(-1)^s}{p^n}((p^n - 1)^s + (-1)^{s+1}).$$

In conclusion, we remark that the finite Fourier series technique may be employed in the case $s = 2$, $m = m_1 = m_2$, to obtain quadratic relations of the Parseval type for sums of the form[9]

$$B(q, r) = \sum_{v=0}^{m-1} (v, q + rv).$$

[1] Eisenstein, G., *J. Reine Angew. Math.*, **27**, 281–282 (1844).

[2] See, for example, Rademacher, H. A., *Am. J. Math.*, **61**, 237–248 (1939). For additional references see Schoenberg, I. J., *Am. Math. Monthly*, **57**, 390–404 (1950).

[3] Davenport, H., *J. Reine Angew. Math.*, **169**, 158–176 (1933).

[4] Vandiver, H. S., these PROCEEDINGS, **33**, 236–242 (1947); **34**, 62–66 (1948); **35**, 681–685 (1949).

[5] Vandiver, H. S., *Ibid.*, **36**, 144–151 (1950).

[6] Faircloth, O. B., and Vandiver H. S., *Ibid.*, **36**, 260–267 (1950). In Lemma I of this paper the condition "$\mu_i \not\equiv 0 \pmod{m_i}$ for any i" may be replaced by the condition "$\mu_i \not\equiv 0 \pmod{m_i}$ for at least one value of i."

[7] See, for example, Davenport, H., and Hasse, H., *J. Reine Angew. Math.*, **172**, 151–174 (1935).

[8] Eisenstein, G., *Ibid.*, **37**, 97–126 (1848).

[9] Dickson, L. E., *Trans. Am. Math. Soc.*, **37**, 363–380 (1935). See also reference 4.

COMPLETE FIELDS FROM LOCAL RINGS

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For a history of the study of topological fields, see Kaplansky's survey of topological rings.[1] Up to the present, no examples of complete topological fields were known except for the fields complete in valuations. The present paper constructs a new class of topological fields which are complete. This class overlaps the class of fields with valuations only in the set of fields with discrete, rank one valuations—which is but a small subset of either class. The new topological fields are constructed from local rings in somewhat the same fashion as fields with non-archimedean valuations are constructed from valuation rings.

Specifically, if R is any integral domain not a field, and F is its quotient field, we may make F into a topological field and hence R into a topological ring by declaring the non-zero ideals of R to be a system of neighborhoods of zero.[2] We shall refer to this as the R-topology. In case R is any ring whose non-zero ideals have a zero intersection, we can define what we shall call the R-topology in R in the same way, except that if $A \cap B = \{0\}$ for two non-zero ideals A and B, we must also allow $\{0\}$ as a neighborhood of zero, getting the discrete topology. For convenience, if R is a field we shall also define the R-topology to be the discrete topology. We may handle all cases simultaneously by referring to the neighborhoods of zero as the open ideals, recalling that this means either all the non-zero ideals or all the ideals, as the case may be. All prefixes R- refer to this topology (R-Cauchy or R-fundamental sequences and directed sets, R-null directed sets, R-limits, R-complete, etc.).

If R is a local ring[3] it has an R-topology, and if R has no zero divisors, its quotient field is even a topological field with continuous division. However, a local ring also has its local topology in which neighborhoods of zero are the powers of the maximal ideal. We shall call this the L-topology. All prefixes L- refer to this topology.

Clearly the R-topology is finer (stronger) than the L-topology. Every R-Cauchy directed set is L-Cauchy; every R-null set is L-null. Hence there is a natural, continuous homomorphism of the R-completion of R into the L-completion of R. The following lemma says this is a continuous isomorphism (not necessarily onto or bicontinuous).

LEMMA. *Let R be a local ring and $\alpha = \{a_\lambda\}$ a directed set in R. If α is R-Cauchy and L-null, then α is R-null.*

Proof. Let A be any open ideal in R. We must show that eventually $a_\lambda \in A$. Since α is R-Cauchy, eventually $a_\lambda - a_\mu \in A$. Fix λ. For suf-

ficiently large μ, $a_\mu \in M^n$ (where M denotes the maximal ideal in R) because α is L-null. Hence $a_\lambda = (a_\lambda - a_\mu) + a_\mu \in A + M^n$. As μ continues to increase, this gives $a_\lambda \in \bigcap_{n=1}^{\infty} A + M^n$. But this intersection is A, which proves the lemma.[4]

THEOREM 1. *Let R be a complete local ring.*[3] *Then R is complete in the R-topology.*

Proof. Let $\{a_\lambda\}$ be a directed set in R which is R-Cauchy. Then it is also L-Cauchy, and since R is L-complete, $\{a_\lambda - a\}$ is L-null for some a in R. But $\{a_\lambda - a\}$ is also R-Cauchy, hence, by the lemma, is R-null. Thus a is an R-limit of $\{a_\lambda\}$.

THEOREM 2. *Let R be a complete local ring without zero divisors. Then the quotient field F of R is complete in the R-topology.*

Proof. Let $\{b_\lambda\}$ be a Cauchy directed set in F. Since R is a neighborhood of zero, $b_\lambda - b_\mu$ is eventually in R. Fixing μ at this high level, $b_\lambda = b_\mu + (b_\lambda - b_\mu)$ becomes the sum of a constant directed set and a directed set in R at least for large λ. But $b_\lambda - b_\mu$ is again R-Cauchy for fixed μ, hence has a limit a in R by Theorem 1. Then b_λ has the limit $a + b_\mu$ in F.

It remains to show that in the situation envisaged in Theorem 2, most of the resultant fields have no valuations preserving their topologies.

THEOREM 3. *If a local ring R has a valuation preserving its R-topology, the valuation is non-archimedean, discrete and of rank one and R is a field or has dimension one.*[3]

Proof. Since R has a valuation it has no zero divisors, hence has a quotient field F. The valuation may be extended to F and will give the same topology in F as the R-topology. Any such valuation will be non-archimedean because F has groups as neighborhoods of zero in the R-topology and hence cannot be a topological subfield of the real or complex field without being discrete; in this case R must be a field. Therefore we may assume we have an exponential valuation V satisfying $V(x + y) \geqq \min [V(x), V(y)]$ with values in an ordered additive group, G. If necessary we may reduce G modulo the isolated subgroup H consisting of all γ satisfying $0 \geqq -|\gamma| \geqq V(r)$ for some r in R. Then V followed by the natural homomorphism on G/H is a new valuation of F giving the same topology as V but having the additional property that R consists solely of elements of non-negative value.

Let B be the valuation ring in F. We have just remarked that we may assume $R \subset B$. Moreover since R is open, $aB \subset R$ for some $a \neq 0$ in R. That is, $R \subset B \subset a^{-1}R$, which proves that B is a finite R-module and is itself a ring with maximum condition. Since the ideals of B are in one-to-one correspondence with the terminal segments in the value group, the positive elements of the value group must be well-ordered. In other words, G is order-isomorphic to the group of ordinary integers, V is a discrete, rank one valuation.

Let P be the maximal ideal in B and Q any non-zero prime ideal in R. Then $P \cap R \subset Q$ because Q, being open in the R-topology (hence in the valuation topology) contains P^k for some k, thus also $Q \supset P^k \cap R \supset (P \cap R)^k$. Since Q is prime, $Q \supset P \cap R$.

Let M be the maximal ideal in R. We show $M = P \cap R$. If not, M contains an element u with $V(u) = 0$. All powers u^s are units in B and so have the property $u^sP^k = P^k$. As above, $M \supset P^k$ so that $M^n \supset u^{n-1}P^k = P^k$, $P^k \subset \bigcap_{n=1}^{\infty} M^n = 0$[5] which implies $P = 0$, the topology is discrete, R is a field, $M = 0 = P \cap R$.

The last two paragraphs imply that if Q is a proper prime ideal in R, then $Q = M$, which proves the theorem.

It follows that a concrete example of a complete topological field with topology not given by a valuation is the quotient field of R where R is the ring of formal power series in n variables ($n \geqq 2$) over any coefficient field —the topology being the R-topology.

[1] Kaplansky, I., "Topological Rings," *Bull. Am. Math. Soc.*, **54,** 809–826 (1948), especially pp. 811–814.

[2] Zelinsky, D., "Topological Characterization of Fields with Valuations," *Ibid.*, **54,** 1145–1150 (1948). Here "topological field" is used to mean a Hausdorff space which is a field in which addition and multiplication are continuous. Division is also continuous if and only if R has a non-zero radical.

[3] For definitions see Cohen, I. S., "On the Structure and Ideal Theory of Complete Local Rings," *Trans. Am. Math. Soc.*, **59,** 54–106 (1946), especially pp. 55 and 58.

[4] Theorem 2 in Krull, W., "Dimensionstheorie in Stellenringen," *J. reine angew. Math.*, **179,** 204–226 (1938).

[5] Krull, W., *loc. cit.*, Theorem 1.

ERRATA

In the article "Continuous Games," These PROCEEDINGS, **37,** 220–223 (1951) on p. 222 in paragraph (1) under THEOREM 3 the words *all absolutely continuous* should be replaced by *an equi integrable set of* so that the third sentence of (1) reads as: Finally, let P represent the cone consisting of an equi integrable set of distributions over the interval (0, 1).

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